Single Numbers, 12/6 net. [No. 41.
free. Volumes commence in JULY. Title and Index
Cases for Binding, 4/- net; or the Volume.
the four numbers, for 7/6 net, post free.

The British Journal of Surgery

UNDER THE DIRECTION OF THE FOLLOWING
EDITORIAL COMMITTEE:

Sir BERKELEY G. A. MOYNIHAN, Bart., K.C.M.G., C.B. (Leeds), *Chairman.*

GEORGE E. ARMSTRONG, C.M.G. (Montreal)
Sir HAMILTON A. BAILLIE, K.B.E., C.B.
Sir GILBERT BARLING, K.C.B., C.B.E. (Birmingham)
ARTHUR W. BURGESS (Manchester)
FREDERIC M. BURGHARD, C.B. (London)
E. POWELL CONNOR, D.S.O., Lt.-Col., I.M.S. (Glasgow)
JOSEPH E. DOBSON (Leeds)
Sir TREVOR CRISP ENGLISH, K.C.M.G. (London)
CHARLES H. FAGGE (London)
JOHN FRASER, M.C. (Edinburgh)
G[illegible] A. GASK, C.M.G., D.S.O. (London)
W[illegible] HANDLEY (London)
[illegible] JOHNSON, O.B.E. (London)
[illegible] JONES, K.B.E., C.B. (Liverpool)
[illegible] KILLINGBECK LANE, C.B. (London)
[illegible] MAC, M.B.E., C.B., D.S.O. (Cardiff)
Sir ALEXANDER MacCORMICK (Sydney)
Sir GEO. H. MAKINS, G.C.M.G., C.B., LL.D. (London)
WM. ERNEST MILES (London)
A. B. MITCHELL, O.B.E. (Belfast)
HENRY A. MOFFAT, D.S.O. (Capetown)
RUTHERFORD MORISON (Newcastle-on-Tyne)
Sir THOMAS MYLES, C.B. (Dublin)
CHARLES A. PANNETT (London)
R. H. PARRY (Glasgow)
Sir D'ARCY POWER, K.B.E. (London)
SIDNEY W. F. RICHARDSON (Capetown)
PERCY SARGENT, C.M.G., D.S.O. (London)
JAMES SHERREN, C.B.E. (London)
GRAHAM S. SIMPSON (Sheffield)
THOMAS SINCLAIR, C.B. (Belfast)
Sir HAROLD J. STILES, K.B.E. (Edinburgh)
HENRY ALEXIS THOMSON, C.M.G. (Edinburgh)
GEO. GREY TURNER (Newcastle-on-Tyne)
GEORGE E. WAUGH (London)
Sir W. I. de COURCY WHEELER (Dublin)
A. W. [illegible] GROVES (Bristol), *Editorial Secretary.*

JULY, 1923

JOHN WRIGHT & SONS LTD.
SIMPKIN, MARSHALL, HAMILTON, KENT & CO. LTD.
MACMILLAN CO. OF CANADA LTD.
THACKER, SPINK, & CO. BOMBAY: W. THACKER & CO.
NEW ZEALAND: WHITCOMBE & TOMBS LTD.
WILLIAM WOOD & CO., NEW YORK, *Sole Agents.*
[illegible] York, N.Y., under the Act of March 3, 879 (Sec. 515, P. L. & R.)

THE

St. Bartholomew's Hospital Operating Table

THIS table, originally designed for the St. Bartholomew's Hospital, is used throughout that institution and in upwards of one hundred other Hospitals in all parts of the country. The table is a composite one and has been built on the suggestion of Mr. H. J. Waring.

A hydraulic oil-pump worked from a pedal enables the heaviest patient to be raised or lowered with but slight exertion. Rotating freely on its base, the table becomes perfectly rigid when clamped. The three ball-bearings permit of its being easily moved from the anæsthetic room to the theatre. The head and leg supports are adjustable to any angle by an automatic lever mechanism operated with one hand.

Other Fittings include :—

Adjustable Back Rest
Shoulder Rests
Skull Rest Arm Rest
Lithotomy Crutches
Anæsthetic Screen
Instrument Tray
adjustable for any position of the arm

Booklet giving full particulars will be sent on application.

Allen & Hanburys Ltd.
48 Wigmore St., London, W.1

THE
BRITISH JOURNAL OF SURGERY

VOL. XI. JULY, 1923. No. 41.

EPONYMS.

BY SIR D'ARCY POWER, K.B.E., LONDON.

IX. POTT'S DISEASE OF THE SPINE.

PERCIVALL POTT published two pamphlets upon that condition of the spinal column which is now called Pott's disease. The first is a small octavo which was printed for J. Johnson, No. 72, St. Paul's Churchyard, in 1779. It is entitled "Remarks on that kind of Palsy of the Lower Limbs which is frequently found to accompany a Curvature of the Spine and is supposed to be caused by it", price one shilling and sixpence. It was translated into French by Dr. A. B. Beerenbroek soon after its appearance, and was published at Brussels. The second pamphlet by the same publisher was issued three years later in 1782 under the title "Farther Remarks on the useless state of the Lower Limbs in consequence of a Curvature of the Spine being a Supplement to a former treatise on that subject". This pamphlet is illustrated by six engravings showing the changes which take place in the vertebræ as a result of the disease.

The first pamphlet may be said to deal with the clinical features of the disease, the second with the morbid anatomy. Pott dedicates his work to Dr. John Lewis Petit, one of the physicians to St. Bartholomew's Hospital, as a small mark of the great esteem and regard of the author. He then goes on to say: "The disease of which I mean to speak, is generally called a palsy, as it consists in a total or partial abolition of the power of using, and sometimes of even moving the lower limbs, in consequence, as is generally supposed, of a curvature of some part of the spine. To this distemper both sexes, and all ages, are equally liable . . . . and until the curvature of the spine has been discovered, it generally passes for a nervous complaint. . . . I have in compliance with custom called the disease a palsy . . . yet there are some essential circumstances in which this affection differs from a common nervous palsy: The legs and thighs are rendered unfit for all the purposes of locomotion and do also lose much of their sensibility, but they have neither the flabby feel, which a truly paralytick limb has, nor have they that seeming looseness at the joints, nor that total incapacity of resistance, which allows the latter to be twisted in almost all

directions; on the contrary the joints have frequently a considerable degree of stiffness, particularly the ancles, by which stiffness the feet of children are generally pointed downward, and they are prevented from setting them flat upon the ground.

"The curvature of the spine, which is supposed to be the cause of this complaint, varies in situation, extent and degree, being either in the neck or back, and sometimes (though very seldom) in the upper part of the loins; sometimes comprehending two vertebræ only, sometimes three, or more . . . but whatever may be the number of vertebræ concerned, or whatever may be the degree or extent of the curvature, the lower limbs only feel the effect —at least I have never once seen the arms affected by it. While the curvature of the spine remains undiscovered or unattended to, the case is generally supposed to be nervous, and medicines so called are most frequently prescribed, together with warm liniments, embrocations, and blisters, to the parts affected; and when the true cause is known, recourse is always had to steel stays, the swing, the screw chair, and other pieces of machinery in order to restore the spine to its true and natural figure; but all, as far as I have observed, to no real or permanent good purpose; the patient becomes unhealthy, and languishing for some time under a variety of complaints, dies in an exhausted, emaciated state".

Pott states that his attention was directed to "this distemper by its occurring in the person of a very promising youth of fourteen years old, with whose family I was nearly connected", and that while the subject was in his mind he happened to be at Worcester "and in a conversation with the late Dr. Cameron of that place I mentioned to him my opinion that previous both to the paralytic state of the legs and to the alteration of the figure of the back-bone, there is a predisposing cause of both consisting in a distempered state of the ligaments and bones, where the curve soon after makes its appearance. The Doctor concurred with me, and said that he remembered some years ago to have noted a passage in Hippocrates in which he speaks of a paralysis of the lower limbs being cured by an abscess in the back or loins. He had acted on this hint and had endeavoured to imitate this act of nature by exciting a discharge near the part and that it had proved very advantageous. A similar good result had been obtained by Mr. Jeffrys, a surgeon of eminence at Worcester. I determined to try the method, and the first case that offered was in an infant whose curvature was in the middle of the neck and who had lost the use of its legs for two or three months. I made an issue by incision on one side of the projection, and gave strict charge to the mother to take care that the pea was kept in; the woman, who had no faith in the remedy, did not take the proper care, and consequently the discharge was not equal to what it should, and might have been; but notwithstanding this neglect, at the end of about three weeks or a month the child was manifestly better and began to make use of its legs; it was then seized with the small-pox and died. My next patient was a tall thin man about thirty-five years old who thought that he had hurt himself by lifting a heavy weight; his legs and thighs were cold and what he called nummy, but not absolutely useless. . . . I made a seton on each side of the curve, and having given his wife directions how to dress them, I called on him once in three or four days. At the end

of six weeks he had recovered the due degree of sensation in his limbs and could rise from his bed and from his chair without assistance. . . . The setons had now, from not having been properly managed, worn their way out, and I would have converted each of them into an issue, but as neither the patient nor his wife had ever believed that the discharge had had any share in his amendment, but on the contrary that he would have been better without it, he would not submit to what I proposed and I left him. At the distance of about three weeks from the time of my leaving him, I met him in the street walking very stoutly, with a common cane, of which he made little or no use. I asked him what he had done; he told me that the sores had continued to discharge till within a few days, but that he had drank a great deal of comfrey-root tea, with isinglas, and he supposed that had cured him. I believe that the cure of this man will, by all who know anything of medicine, be thought to be so unlikely to have been affected by the comfrey and isinglas that my inference in favour of the seton will not be thought unreasonable, and that my determination to prosecute the method, from what I had seen and heard, was well founded ".

In spite of these discouragements Pott continued the treatment, and " within these last six or eight months several cases of curved spine have been received into St. Bartholomew's Hospital, where they have been seen by great numbers of the profession. The novelty of the treatment and the success which has hitherto constantly attended it has necessarily engaged the attention of many and occasioned some observations on the subject. . . . Although I have called this an early publication, yet I have waited a sufficient length of time and have treated a sufficient number of subjects to be clear in the truth of what I have asserted as far as such time and such individuals go. That the patients whom I have attended in the early part of the distemper of whatever age, have all got well; that is have not only regained the use of their legs but have become healthy, and fit for any exercise or labour, as numbers can testify who have seen them daily. Most of them have become much straiter, some quite strait, and all of them perfectly free from all kind of inconvenience arising from the curve. . . . That as far as my experience goes I have not the least doubt, that if the means proposed, be made use of before the bones become really carious and rotten, that they will always be successful. When indeed a truly rotten state of the bones takes place no good is to be expected from this or from anything else, but it should be observed at the same time, that this never happens but when the distemper is of very old date, and that when this is the case, the whole machine is so disordered, and the patient so truly and so generally distempered, that there can be no reasonable expectation of success from any thing ".

There is no doubt that Pott was perfectly honest in his belief, but the whole train of argument shows that he was relying entirely upon empiricism, and that, having a firm belief in the efficacy of counter-irritation, he made no effort to seek for any other cause for the improvement. He did not take into consideration the curative effects of rest whilst the patients were in hospital, nor did he make any careful post-mortem examinations of the diseased tissues, for he contented himself with a mere examination of the vertebral column and with maceration of the bones. In excuse it may be urged that

John Hunter was one of his pupils, and consequently surgical morbid anatomy was not yet existent.

Pott begins his "Farther Remarks" with the following words: "It is now near three years since I first troubled the public with my observations on the disease which makes the subject of the following tract". He excuses his former paper on the ground of the importance of the subject, the perfect safety of the experiment, and the desire to obtain evidence of the utility of his method of treatment from other surgeons. "My wishes and my expectations have been fulfilled. I have received such manifold and repeated testimony of the success of the proposed method, from so large a number of the most eminent practitioners, not only in this town and kingdom, but in many other parts of Europe; that these, added to my own experience, have completely satisfied me, and enabled me to say, that in proper cases, and under proper treatment, I have no doubt of its being universal. In all the time which has passed since the first publication, I have sought and embraced every opportunity of obtaining information, both from the living and from the dead". He then recapitulates what he had stated in the previous communication and gives an excellent picture of the early symptoms: "The account most frequently given is, that for some time previous to the incapacity, the child had been observed to be languid, listless, and very soon tired; that he was unwilling to move much or briskly; that he had been observed frequently to trip and stumble, although no impediment lay in his way; that when he moved hastily or unguardedly, his legs would cross each other involuntarily, by which he was often and suddenly thrown down; that if he endeavoured to stand still, and upright, unsupported by another person, his knees would totter and bend under him; that he could not with any degree of precision or certainty, steadily direct either of his feet to any particular point, but that in attempting so to do, they would be suddenly and involuntarily brought across each other; that soon after this, he complained of frequent pains and twitchings in his thighs, particularly when in bed, and of an uneasy sensation at the pit of his stomach; that when he sat on a chair, or a stool, his legs were almost always found across each other, and drawn up under the seat; and that in a little time after these particulars had been observed, he totally lost the power of walking".

The picture seems very simple, and it could now have been drawn by any observant medical student, but it had never been painted before, and it has now become a part of surgery. He continues: "In infants the curve is seldom noticed till it has got to such a size and state, as to demand attention from the deformity; previous to this, all the marks of distemper which appear in the child, pass for the effects of general weakness, and are treated as such; differently by different people, and under different circumstances, but never with any permanent good effect; some of the adventitious symptoms, if I may so call them, are in some degree relieved, but the principal remain in full force, or what is much more frequent, go on increasing. In an adult it passes for rheumatism, or gravel, or a sprain, and the defect in the limbs is the first thing that occasions an enquiry into the state of the back bone. The true cause of the disease is a morbid state of the spine, and of some of the parts connected with it; which distempered state of parts will, upon careful

Plate 6.

A View of some of the Vertebra in a case of Curved Spine which had been Cured by the Cautec. & which were taken from the body of the Patient who died of another distemper at some distance of time after. In this may be seen the State of the Vertebra which had been Crushed, and of the Consequent Anchylosis or Union.

R. Laurie, fc.

Act. 10. 1782. Published as the Act directs. by J. Johnson London.

enquiry, be always found to have preceded the deformity some length of time; in infants this is the sole cause and external violence has nothing to do with it. In the adult, I will not assert that external mischief is always and totally out of the question, but I will venture to affirm what is equal, as far as regards the true nature of the case, which is, that although accident and violence may in some few instances be allowed to have contributed to its more immediate appearance, yet the part in which it shows itself, must have been previously in a morbid state, and thereby predisposed for the production of it.

"The primary and sole cause of all the mischief, is a distempered state of the parts composing or in immediate connection with the spine, tending to, and most frequently ending in a caries of the body, or bodies, of one or more of the vertebræ; from this proceed all the ills whether general, or local, apparent, or concealed; this causes the ill health of the patient, and, in time, the curvature. The helpless state of the limbs is only one consequence of several proceeding from the same cause; but though this effect is a very frequent one, and always affects the limbs in nearly the same manner; yet the disease not having its origin in them, no application made to them only can ever be of any possible use. The same failure of success attends the use of the different pieces of machinery, and for reasons which are equally obvious.

"They are all, from the most simple to the most complex, but particularly the swing and the screw, calculated to obviate and remove what does not exist. They are founded upon the supposition of an actual *dislocation* which never is the case, and therefore they always have been and ever must be unsuccessful."

Pott then reiterates his belief in the value of counter-irritation in the treatment of these cases, saying, "It is a matter of very little importance towards the cure, by what means the discharge be procured, provided it be large, that it come from a sufficient depth, and that it be continued for a sufficient length of time. I have tried the different means of setons, issues by incision, and issues by caustic, and have found the last in general preferable, being least painful, most cleanly, most easily manageable, and capable of being longest continued. The caustics should be applied on each side of the curvature, in such a manner as to leave the portion of skin covering the spinal processes of the protruding bones, entire and unhurt, and so large, that the sores upon the separation of the eschars, may easily hold each three or four peas in the case of the smallest curvature; but in large curves, at least as many more. These issues should not only be kept open, but the discharge from them should be maintained by means of orange peas, cantharides in fine powder, aerugo aeris, or any such application as may best serve the intended purpose, which should be that of a large and long-continued drain".

Having considered the clinical and curative aspects of the disease, Pott concludes these remarkable contributions to surgery by some remarks upon the morbid anatomy of the condition, saying: "This morbid affection shews itself in a variety of forms, but although its appearances be various, yet they are always such as determine the true nature of the distemper. Sometimes it appears in a thickened state of the ligaments, connecting the vertebræ together, without any apparent affection of the bones. Sometimes in the

form of a distempered state of the intervertebral substances, called cartilages. Sometimes in that of diseased glands, either in a merely indurated and enlarged state, or what is more frequent, in that of a partial suppuration. Sometimes it is found in the form of bags or cysts, containing a quantity of stuff of a unequal consistence, partly purulent, partly sanious, and partly a curd-like kind of substance; and not unfrequently entirely of the last. Sometimes under these bags, or cysts, even while they remain whole, the subjacent bones are found to be distempered, that is deprived of periosteum, and tending to become carious. Sometimes these collections erode the containing membranes, and make their way downward by the side of the psoas muscle, towards the groin, or by the side of the pelvis behind the great trochanter, or in some cases to the outside of the upper part of the thigh. The disease which produces these effects on the spine, and the parts in its vicinity, is what is in general called the scrophula; that is, that same kind of indisposition as occasions the thick upper lip, the tedious obstinate ophthalmy, the indurated glands under the chin, and in the neck, the obstructed mesentery, the hard dry cough, the glairy swellings of the wrist and ancles, the thickened ligaments of the joints, the enlargement, and caries of the bones, &c., &c., &c.".

Both essays are well worth reading, as well for their excellence as for their defects. They show Pott to have been better in differential diagnosis and treatment than in his knowledge of morbid anatomy. But considering the difficulty of making post-mortem examinations in the " dead house beneath the Cutting Ward at St. Bartholomew's Hospital ", the specimens he succeeded in obtaining were satisfactory. It is clear that the results of the treatment he recommended were due to the enforced rest; but the doctrine of physiological rest was not enunciated for nearly one hundred years after his death. He carried it out empirically, and in so doing displaced for ever the ambulatory system and the use of the complicated mechanical apparatus which had been a torture to many unhappy patients.

The illustration is reproduced from the rare original pamphlet, and shows the stains or foxing which are not uncommon in paper of the period.

THE FLOW OF LYMPH FROM THE ILEOCÆCAL ANGLE, AND ITS POSSIBLE BEARING ON THE CAUSE OF DUODENAL AND GASTRIC ULCER.

By L. R. BRAITHWAITE, Leeds.

(Being the Arris and Gale Lecture delivered at the Royal College of Surgeons of England on February 21, 1923.)

GENERAL OBSERVATIONS.

Evidence of inflammatory change in the right iliac fossa is common enough to be familiar to every surgeon. There is no part of the intestinal canal where such remarkable adhesions, bands, and kinks arise. The gall-bladder frequently presents striking adhesions, and to a much less extent the splenic flexure of the colon (Payr's disease). According to Lane, there are non-inflammatory reasons for the ileal kink and for Jackson's cæcal membrane; but it is frequently obvious that exactly similar conditions appear as a result of some pathological process usually arising in the appendix. It is common enough to see the various adhesions, bands, and kinks actually in the stage as they are laid down by deposits of plastic lymph, adhesions which can easily be separated by the finger. It is not possible to escape from a belief that for the most part these conditions are inflammatory. Certainly disease of the appendix is the almost universal cause, though conditions arising in the cæcum and lowest part of the small intestine probably sometimes play a part (*Fig.* 1).

Fig. 1.—Showing the common types of adhesions found in the pericæcal area.

Subacute inflammation of the appendix is accompanied by a widespread peritonitis, more marked in its virulence and extent than that due to inflammation of the rest of the alimentary canal. In cases where no history of an acute attack of appendicitis can be obtained, bands of omentum

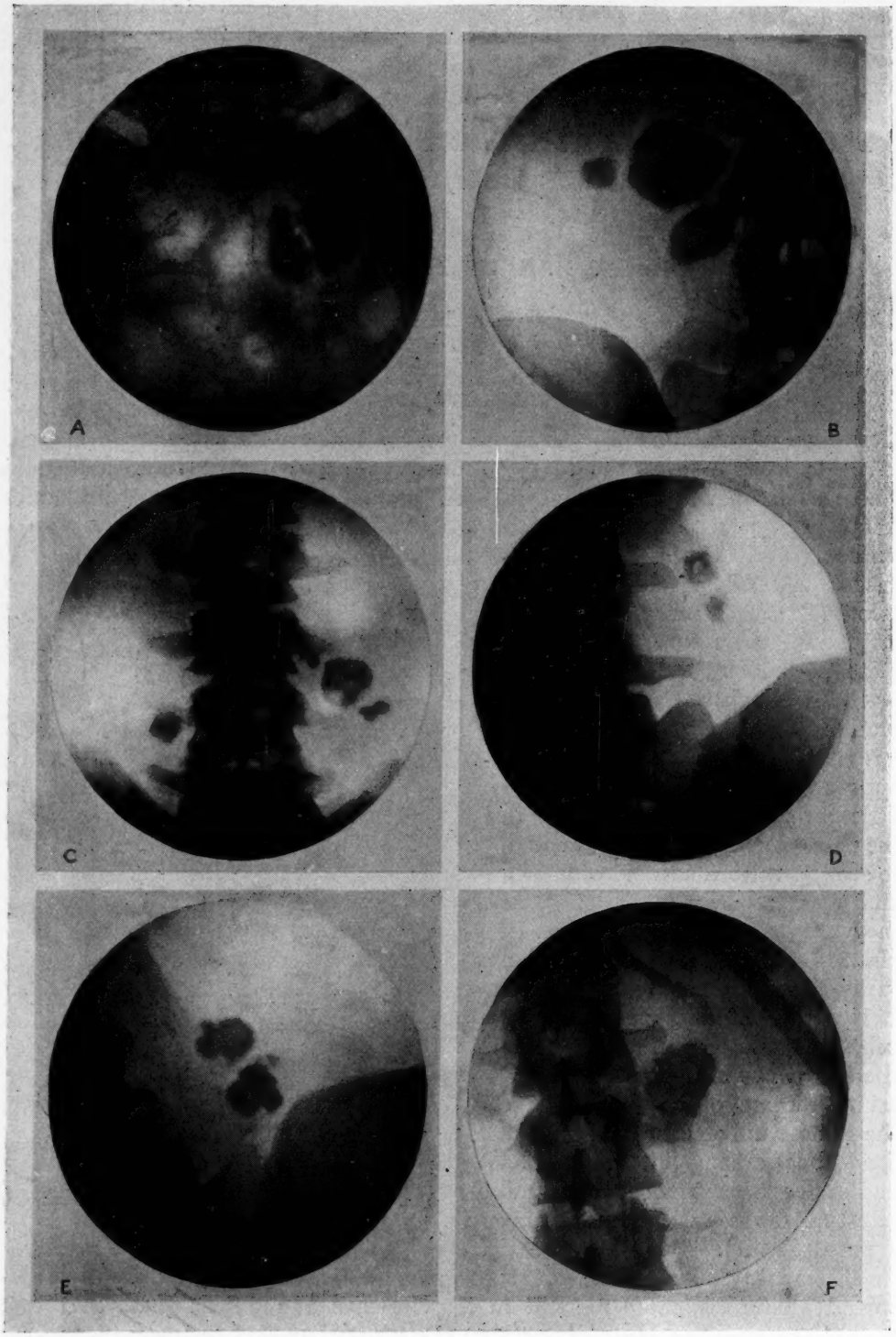


FIG. 2.—Radiograms of calcified ileocæcal glands (*all except* B *are shown reversed*).

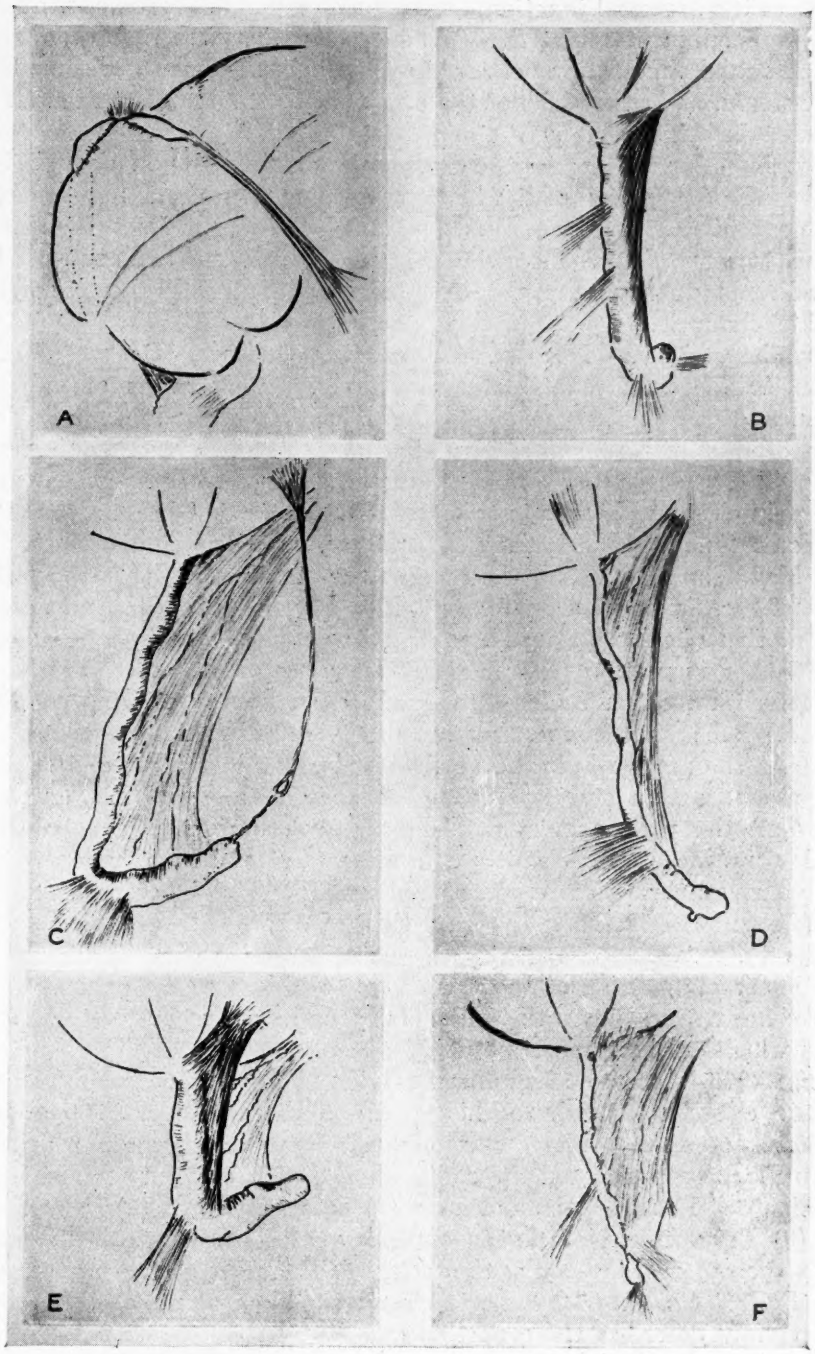


FIG. 3.—Selected drawings of the appendix as seen in cases of duodenal ulcer.

crossing the ascending colon, and kinking of the ileum, one, two, or three inches from the ileocæcal junction, are frequently found, together with a badly diseased appendix; it might fairly be suggested therefore that there is some faculty of actual penetration possessed by the infecting agent, or some tenuity or other weakness in the walls of the appendix or the bowel contiguous to it, not present in other parts of the alimentary canal.

That infection arising in the ileocæcal area is common enough is also shown by the frequency with which both tuberculous and non-tuberculous enlargement of the ileocæcal glands is found (*Fig.* 2). Does this infection give rise to any disturbances in other parts of the abdomen? Under the conditions usually seen, infection is clearly most likely to spread by lymphatic channels.

Moynihan, Mayo, Murphy, MacCarthy, Ochsner, Deaver, McGrath, Paterson, Wilkie, and others have considered certain kinds of indigestion, duodenal and gastric ulcer, and gall-stones as possibly due in some way to the changes seen in the region of the appendix. In an epoch-making article, "Appendix Dyspepsia", Moynihan[1] describes cases of dyspepsia associated with certain obvious changes in the stomach. Paterson,[2] in an article written at the same time and published slightly later, writes on similar lines.

In Moynihan's words, in these cases the stomach presents the following signs: "There is no thickening, no whiteness, no puckering, no adhesions. The stomach looks in every particular quite normal. But if it be allowed to lie quietly for inspection (and it is better to watch it while the abdominal wall is raised up, before the organ is handled), a most interesting condition is displayed. The stomach in its pyloric half is seen to be in vigorous and excited action. At the point where the vertical and horizontal parts of the stomach merge, a contraction starts and spreads towards the pylorus, and at last involves all the pyloric antrum. The stomach becomes thick, contracted, and pale; its muscle is evidently in a state of strained and vigorous action, and the channel through it is almost obliterated. On the cardiac side of this area of spasm the stomach is quiet, a little distended even, and shows no movement. . . ."

In his second edition of *Duodenal Ulcer*[3] will be found a page of drawings made by the writer, illustrating the conditions in the region of the appendix, some of which are here reproduced (*Fig.* 3).

Since 1908, those who have been privileged to work with Moynihan have given the closest attention to this condition through over 1000 abdominal operations, and have come to recognize three gastric stigmata which enable them to diagnose a diseased appendix almost with certainty whilst it is still out of sight. The three stigmata are: (1) Pyloric spasm; (2) Pyloric congestion; (3) Enlargement of glands on the greater curvature of the stomach towards the pylorus, i.e., along the course of the right gastro-epiploic vessels.

1. *Pyloric Spasm.*—Moynihan's description has already been repeated; it still vividly describes the condition (*see Fig.* 4).

2. *Pyloric Congestion* is a term which allows of wide limits: by it is indicated a blushing of this part of the stomach which is not normally present—it can only be seen in the intervals between the spasms; and because the surface vascularity of the normal stomach is greatest towards the pylorus,

it is only as a result of constant practice in abdominal surgery that one feels competent to appreciate it.

3. *Enlargement of the Glands.*—This is in another category ; it is easily seen *if looked for*, but is frequently obscured by fat. The glands lie between the

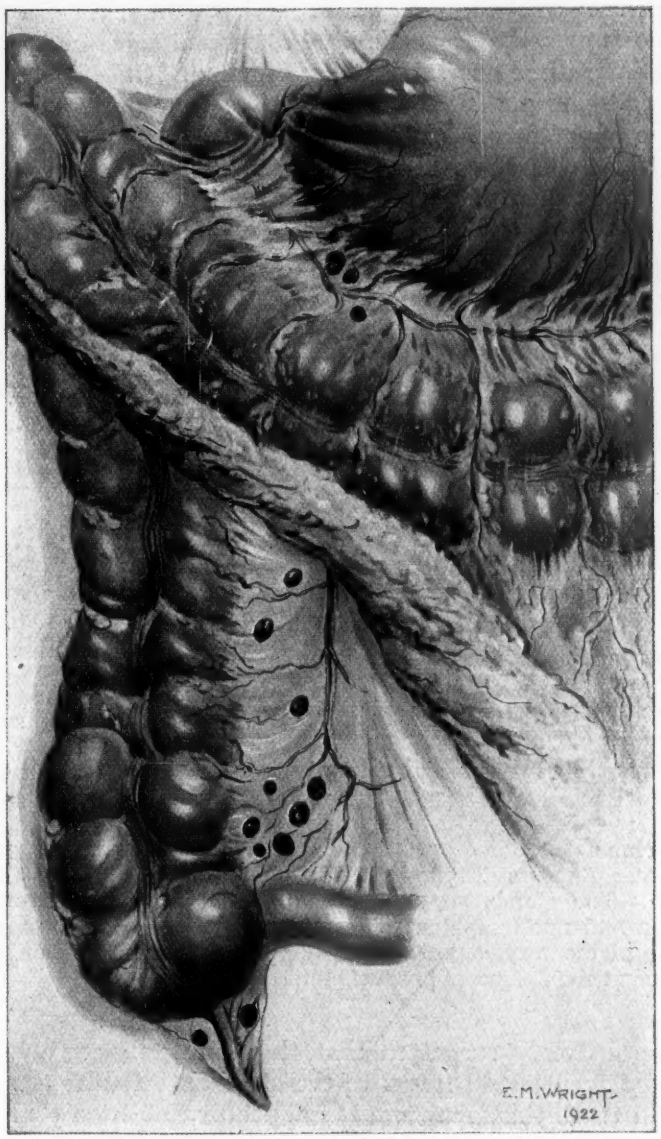


FIG. 4.—Showing pyloric congestion and spasm, also the distribution of pigment in the case of ' pigmented appendix '.

layers of the great omentum. They are rarely close to the pylorus—more often two to three inches away. As a rule they are in two groups : the upper group consists of one or two glands lying above or on the right gastro-epiploic vessels ; the lower and more numerous group always lies below the

vessels. It is a point of some importance that in these cases the lower group only is enlarged, as a rule. The glands are always red, fleshy, and freely movable.

In 1914 the writer came across a case which is remarkable in itself, and of interest in that it led him to commence investigations into the causes of the enlargement of this group of glands. A lady, 30 years of age, was sent in with a diagnosis of acute appendicitis. The usual signs and symptoms were present, and immediate operation was performed. On opening the abdomen an astonishing sight presented itself. The appendix was slightly congested but otherwise apparently normal. The ileocæcal glands were jet-black, and a chain of jet-black glands could be traced up to the duodenum and the superior mesenteric vessels where they cross it. In addition, two of the glands on the greater curvature of the stomach, two inches from the pylorus, were black (*Fig.* 4). The appendix was removed, one gland from the ileocæcal angle, and one from the stomach group. The pathologist's report was as follows:—

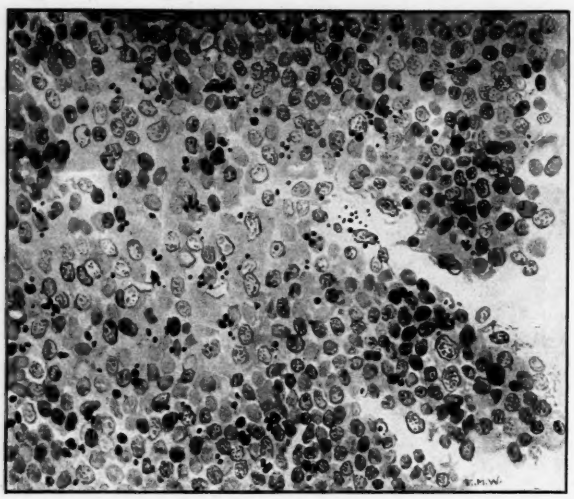


FIG. 5.—Microscopic appearance of lymph gland containing pigment derived from appendix.

REPORT ON APPENDIX AND GLANDS.

1. *Appendix.*—There is no evidence of malignant disease. The proximal third of the appendix mucosa is deeply pigmented. This is not a very rare finding apparently. Microscopic examination shows masses of pigment inside phagocytic cells disseminated in the interglandular tissue and following the vascular channels. Some parts have collected at the periphery of the follicles of the lymphatic glands still lying within the endothelial cells.

2. *Glands.*—The glands supplied are abnormal in their central sinuses, containing plasma and not lymph, as if this were a form of hæmolymph-gland. The reason for the pigmentary deposit is the appendical lumen. (Change in the fæces, with absorption of the abnormal products ?). (*Fig.* 5.)

O. C. GRUNER.

This case certainly suggested that there must be some lymphatic path whereby the pigment could travel to the stomach. It also suggested that, if pigment could reach this area from the appendix, infection could do the same thing.

NORMAL FLOW OF LYMPH.

The normal flow of lymph from the ileocæcal angle in a perfectly healthy individual, as gleaned from post-mortem injections—largely in the fœtus—is clearly defined. Gathering tributaries from the appendix, the cæcum, and the last six inches of the small intestine (in part only), the lymphatics pass

irregularly in a group of five or six vessels to the chain of glands which lies dotted along the line of the ileocolic artery. The main efferent trunks pass up in front of the third part of the duodenum, reaching the group of glands massed round the superior mesenteric artery, and so the lumbar chain. If, now, it be true that from here the efferents pass wholly and directly to the lumbar group and so to the receptaculum chyli, then there can be no direct path of lymph from the ileocæcal angle either to the stomach or the duodenum. It was the object of this research, by experiments in both living and dead subjects, to find out: (1) Whether aberrant paths existed; and (2) What effect disease of lymph vessels or glands would have on the lymph flow.

Experiments in Animals.—For this purpose experimental work was commenced on cats. The various lymph-sheds of the abdomen were injected by means of a hand syringe with a very fine steel needle. All cats would appear, on abdominal section, to have masses of diseased mesenteric glands; most of these on microscopical examination prove to be quite healthy, some prove to be tuberculous. The diagnosis as between a hyperplasia of a lymph gland and an early condition of tuberculosis is not easy even with the microscope. It was found that there were vast differences between the living and the dead animals, both in the ease with which the injection could pass through lymphatic glands and vessels, and in the extent of area injected. The arrangement of the intestinal canal of a cat is primitive; the appendix is weakly represented by a rather marked cæcal tip, which, however, contains large masses of lymphoid tissue.

After considerable practice to obtain a steady and even pressure—by no means an easy business—it was found to be possible, in living cats where the glands were not obviously diseased, to inject indigo-carmine from the tip of the cæcum into the whole lymph-shed up to the pancreas and over it into the glands which lie *above* it. Glands which lie in close contact with the upper and lower border of the pylorus were frequently injected from the cæcal wall. In the dead animal (many attempts were made very soon after the animal had been taken from the lethal chamber) this was never accomplished; the injection would reach the lower border of the pancreas, but no further.

An attempt was now made, by implanting active tuberculous gland tissue from a human being into the submucous layer of the cæcal wall of a cat and into the wall of the small intestine near the ileocæcal valve, to produce in course of time a tuberculous manifestation in the pyloric glands. Eventually, starting with an animal whose glands appeared healthy and which were healthy on microscopic examination, a chain of tuberculous glands was set up in the ileocæcal angle, leading to a tuberculous gland in the subpyloric line. A kitten taken early from its mother and fed for three months on sterile food was found to have healthy mesenteric glands; after being fed on tuberculous gland tissue from human necks, together with other and *sterile* food, it developed tuberculosis of the mesenteric and subpyloric glands. These specimens, together with the gland sections, were shown at the meeting of the Association of Surgeons in May, 1922.

Experiments in Man.—It was argued that, as indigo-carmine is so extensively used in human beings for the purpose of estimating the work done by the kidneys, and is passed from a subcutaneous or intramuscular injection

through the organism without any ill effects, it could not inflict any injury if injected into the lymph system of the appendix or other abdominal organ for the purpose of estimating whether or not the same striking ease and speed of flow as was seen in the living cat would be seen in the living man. Advantage was taken of unusual opportunities for performing abdominal section in man, to attempt similar injections to those made in animals. For this purpose a more equable and a definitely-known pressure was achieved by the use of a mercury column raised by air-pressure from an ordinary bicycle pump (*Fig.* 6). The finest steel needles were used, fixed into thick rubber tubing, and a pressure of from 6 to 8 mm. of mercury was found to be about sufficient; the pressure was increased where obstruction to the flow was observed. A careful sterilization of the storage chamber and the efferent tubes was effected, the latter being boiled before each injection. The solution used was similar to that injected into animals, i.e., 1–60 indigo-carmine in distilled water. There were no ill effects either accompanying or following the injections, and as there was no mortality there are no specimens to show. Forty-eight injections were made in all.

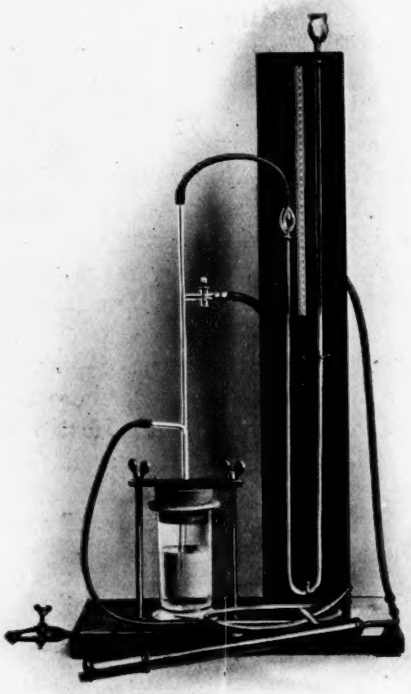


FIG. 6.—Mercury column raised by air pressure from a bicycle pump.

What is already known of the difference in the ease of injection of healthy lymphatics under varying conditions?

Hunter was the first to point out that the difficulty of injecting lymphatics increased rapidly each hour after death, and also that injection was easier in young than in old subjects. Every worker has noted similar variations. Cuneo and Delamere,[4] in their work on the lymphatics of the stomach, remark that "the stomach used should be as fresh as possible"; they regard injection of the subserous reticula as "almost impossible so soon as the peritoneal coat has lost its normal consistency". They recommend that the stomachs of newly-born or very young subjects only be used. Moynihan,[5] in connection with the lymphatic system of the stomach, says, "The greater part of the work of investigation has been carried out upon the bodies of fœtuses or infants", and, "It is stated by Polya and von Navratil that the number of the glands increases considerably in adult life either by the division of the original glands or by their fresh development from lymph vessels". Dobson and Jamieson[6] carried out their work in fœtuses or the newly-born.

Deposits of fat in the mesentery always prevented any injection being attempted, because both glands and vessels were obscured.

One striking feature of an injection into the submucous coat of the stomach in the living subject as compared with a similar injection in the dead under a similar pressure is shown in the diagram (*Fig.* 7). It will be noticed that the area coloured by the injection in the living stomach is one and a half times to twice that coloured in the dead organ; in addition, the former injection takes place much more quickly than the latter.

The first series of injections was made into the appendix. It proved everything Jamieson and Dobson and others have written with regard to the flow of lymph from the ileocæcal angle; no communication was found with the lymphatics of the pelvis, but a few vessels were found to pass into the retroperitoneal space of the right iliac fossa. It was found repeatedly, however, that an injection into the appendix would send the dye inwards to the

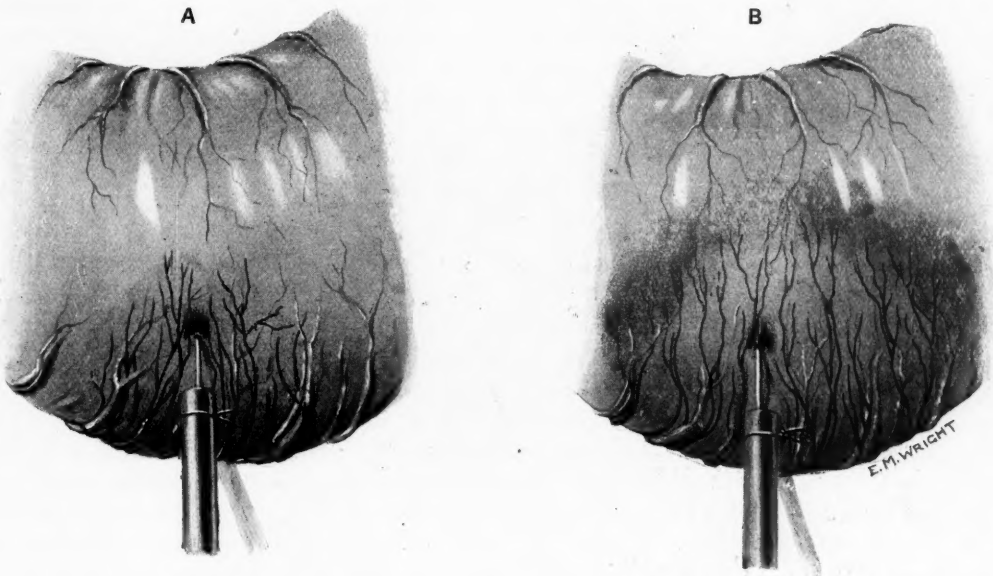


FIG. 7.—A shows area of stomach wall injected in dead, and B in living subject under similar pressure.

small intestine and, at a higher level, outwards to the ascending colon (aberrant and retrograde flow). On rare occasions an appendical injection, passing through gland after gland in the ileocæcal chain, would fill the group of glands round the trunk of the superior mesenteric artery, but no more; after frequent injections into the appendix it was noted that injection into one of the ileocæcal glands was a more certain method of colouring this group. The entry and exit of lymph to and from the glands round the trunk of the superior mesenteric artery where it crosses the third part of the duodenum being the immediate object of the inquiry, this method of accomplishing their injection was very largely used.

It may be said at once that injection of the glands on the greater curvature of the stomach near the pylorus (Jamieson and Dobson's 'right gastro-epiploic group') was never achieved through the ileocæcal chain, though, as we shall

see later, it was achieved in another way. It should, however, be understood that connecting links—though in theory they are efferent paths—do in fact exist. Poirier and Charpy[7] describe not only close connection between the glands on the greater curvature near the pylorus and the group of glands which lies in the angle between the first and second parts of the duodenum, in front of the head of the pancreas (Jamieson and Dobson's 'subpyloric group'), but also a further path along the right gastro-epiploic vein directly to the superior mesenteric group.

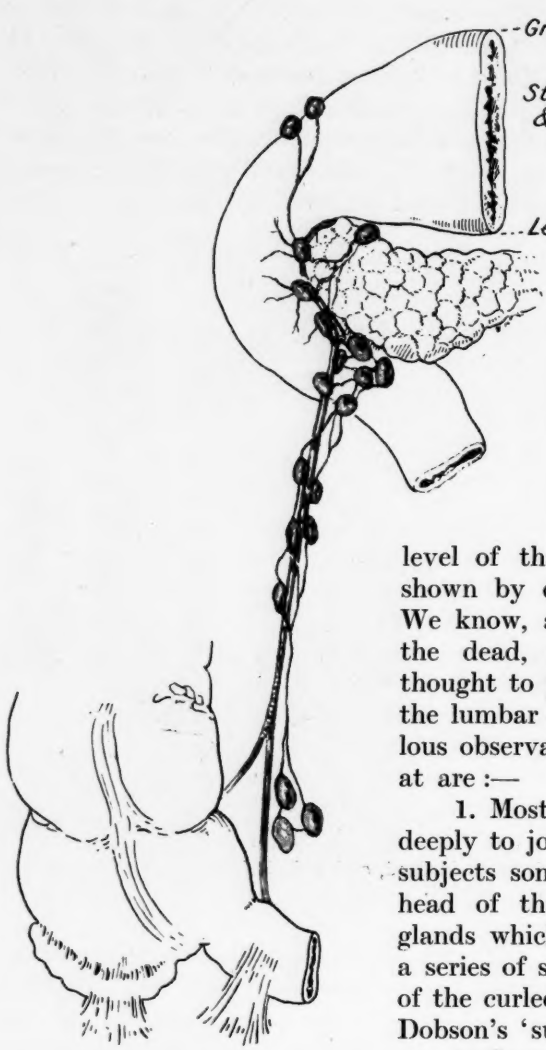


FIG. 8.—Diagram showing area of glands injected in the living subject from an ileocæcal gland puncture.

The great object of the inquiry was to find out what is the path of lymph *after* it has reached the level of the third part of the duodenum, as shown by experimental injection in the living. We know, as a result of very careful work in the dead, that the whole of the lymph is thought to pass below the pancreas straight to the lumbar chain. After prolonged and scrupulous observation the general conclusions arrived at are :—

1. Most of the lymph undoubtedly passes deeply to join the lumbar group ; but in living subjects some is seen to pass upwards *over* the head of the pancreas to enter that group of glands which frequently lies in a crescent or in a series of small groups along the inner border of the curled duodenum (*Fig.* 8) (Jamieson and Dobson's 'subpyloric group').

2. Some undoubtedly passes *through this group on to the duodenal wall itself and up to, and occasionally beyond, the pylorus.* The injection has been seen on two occasions to pass even higher, reaching the chain of glands along the common bile-duct, and even colouring the cystic gland.

Post-mortem Experiments.—Experiments were now made in young human subjects, post mortem, by the use of Gerota's Prussian blue and the mercury pump (8 to 10 mm. Hg), an attempt being made to reproduce the injections already accomplished in the living. They were not nearly so uniformly successful.

FIG. 9.—Drawing of an injected post-mortem specimen (the needle was placed in an ileocæcal gland). Retrograde flow back to the cæcal wall is clearly shown.

Fig. 9 shows injection of the crescentic group of glands on the head of the pancreas near the duodenum quite well, though in the living subject they are more clearly marked; they were made into an ileocæcal gland. The line of injection appears to follow fairly accurately the arc made by anastomosis of the superior and inferior pancreaticoduodenal vessels, though a much more irregular distribution is common. It would therefore appear possible to inject, both in the living and the dead, an area much wider in extent than would be expected from a study of the 'normal' anatomical paths apparently followed by the lymph flow.

Can the discrepancy between these experimental findings and the usual text-book description of lymph flow in this area be accounted for by an unusual success on the part of the investigator in his injections in the dead and living subject, opening up paths hitherto not known? It is certain that, although the flow of lymph is very adequately described by many writers as from the periphery of each great lymph-shed to its termination in one or other of the big groups of glands on and near the head of the pancreas, the possibility of connections existing between the termini themselves has never been investigated. It is possible that there are in existence reserve sets of lymph channels as well as lymph glands, which come into play: (1) Under unusual pressure of lymph flow; and (2) In the presence of obstruction in the normal channels, due to disease.

Stiles[8] describes very minute glands, 1 to 2 mm. in diameter, possessing a very primitive structure and consisting of a single lymphoid follicle connected with one afferent and one efferent lymphatic, existing normally in the axilla, which are not described as occurring amongst the usual axillary glands. He thinks them capable of becoming properly developed glands and of taking the place of glands removed by operation or put out of action by disease. Such a possibility—also dwelt upon by C. H. Mayo—is of considerable interest and importance in considering the question of aberrant and retrograde lymph-flow in the presence of long-standing infection.

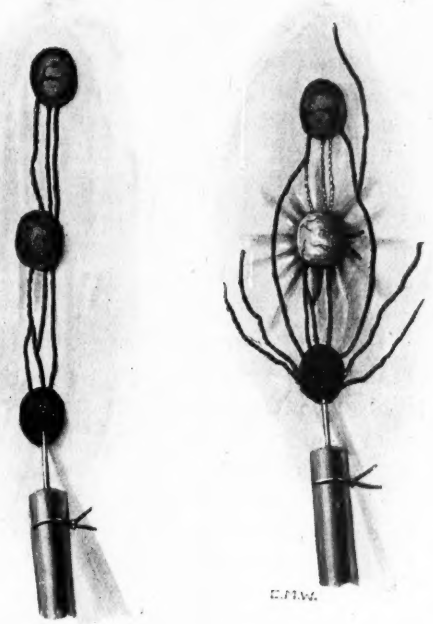
THE EFFECT OF DISEASED GLANDS ON THE LYMPH FLOW.

Experiments were made in the living subject on the effect of diseased glands on lymph flow. A lymph gland was drawn up from the mesenteric surface, and its base was very tightly ligatured; the ligature exerted a similar effect to that produced by a chronic lymphadenitis (*Fig.* 10). In every experiment in the living subject, injection made into the gland next below the ligatured gland failed to enter the ligatured gland, and after a moment's hesitation a new sheaf of aberrant vessels came into play, which carried the injection fluid round the ligatured gland to the gland next but one or more above it; in addition, the injection sometimes flowed backwards towards the gland below. We produced, therefore, a retrograde as well as an aberrant flow of lymph. In four cases, where an obvious old tuberculous gland was found, injection was made into the next healthy gland below, of the same chain. In all the cases it was clear that the diseased gland acted as an obstruction; sometimes there was partial permeation of the gland, its surface presenting a chessboard pattern of blue and white squares; sometimes an

efferent vessel would become injected in addition to one or two afferents; sometimes there was no flow whatever into any area within one-half to one inch of its periphery, the afferent vessels appearing to stop at a blind end. It was never found possible to reproduce these experiments post mortem.

Cornet's law of localization lays down that infection—in a previously healthy lymphatic system—within lymph channels, always travels from one regional group of glands to the next regional group in the direction of the lymph flow. Retrograde flow of lymph in a healthy subject is denied as a possibility by most writers, and probably this is true to a very great extent; but it is certain that it may take place, and was, as is seen, often noted in the course of the experiments even in apparently healthy subjects.

Most, of Breslau,[9] says that retrograde flow can only occur when there is serious disturbance of circulation; he illustrates retrograde flow in a coloured drawing, as occurring from a septic sore on the leg, the lymphangitis spreading chiefly up the leg, but also for some inches downwards.



A B

FIG. 10.—Shows (A) the normal injection of a chain of glands, and (B) the effect of ligation of the middle gland.

In Moynihan's book, *Abdominal Operations*,[5] he states: "In cases of cancer (stomach) the early involvement of certain lymph vessels, their plugging by cancer-cells, or the implication of a single gland, may be enough to disturb the normal direction of the lymph current." Sampson Handley has shown that metastases from a carcinoma of the breast may pass down to the rectus sheath and so to the liver. He has also reported that carcinomatous cells from a rectal growth can be found six or eight inches below the primary growth, though this is denied by later workers (Cole, Monsarrat and Williams, and Cheatle). Every surgeon must know the frequency with which the glands along the common bile-duct become enlarged in cases of carcinoma of the stomach; this can only come about by retrograde lymph flow. Is it not more than possible that secondary carcinoma in the liver arising from both stomach and intestinal canal may have passed along lymphatic vessels by retrograde flow?

McVay[9] says rectal carcinoma may reach the liver through glandular metastasis or by breaking off of emboli of carcinoma-cells into the portal circulation. Joseph Wiener[10] considers it possible that the ileocæcum may be secondarily infected by retrograde flow from diseased mesenteric glands. Broders,[11] describing a case of gastric tuberculosis, quotes Claytor and

Wilkinson as considering that tuberculosis may affect the stomach from adjacent tuberculous glands. He also states that Chiari had a case of a similar kind. Rosset thought his case of tuberculosis of the stomach was due to a primary focus in the lymph glands at the hilum of the left lung, and that it infected the retrogastric lymph glands and then the stomach by a blockade of the lymph stream. The late Edward Ward, of Leeds, used to illustrate the very free collateral circulation of lymph and the presence of aberrant flow as a result of disease by the condition seen in cases of malignant disease developing in a long-standing lupus (usually facial). Owing to the sclerosing of normal lymph channels by tuberculosis, the superadded malignant disease is unable to reach the lymph glands, and they do not, in fact, become enlarged. As soon as the malignant disease reaches healthy skin, the lymph nodes become affected, sometimes on the same side of the neck, but frequently on the opposite side.

Last month a lady was seen in consultation with Dr. Hall. She presented an interesting picture of retrograde lymph flow. As a V.A.D. during the war she had suffered repeatedly from septic fingers, chiefly in the left hand, and presumably from lymphangitis and lymphadenitis. She was recently vaccinated in the usual place in the upper part of the left arm, and after ten days developed acute suppuration in the tendon sheaths in front of the wrist of the same side. There was no appreciable lymphadenitis in the axilla, and no sign of any septic focus except the vaccination spots.

Most[12] suggests that a tumour forms in growing new lymphatic sheds with new channels for evacuation. Thus it is possible for new glandular regions to be infected by way of these new vessels (aberrant flow). Gerota himself, in a case of carcinoma of the breast with metastases in the inguinal glands, determined, by actual injection at the post-mortem, the newly-formed lymphatic tract.

It might be argued that to illustrate retrograde lymph flow by the spread of carcinoma is not a fair thing, because it is not so much a flow as a permeation. It is true that at the edges of a carcinoma lymph channels are seen blocked by an unbroken line of malignant cells; but Sampson Handley and others have shown that at a distance from their source the cells lie in the lymph channels in solitary masses with stretches of normal lymph vessel intervening. This would certainly suggest that both a permeation and a flow are present: first a permeation, then a 'wasting away' of the outlying cells by the fluid lymph.

We shall see later that, assuming the text-book description of lymph flow to be correct, the view that there is a retrograde or aberrant flow of lymph must be accepted before any reasonable theory of the existence of a lymphatic flow from the ileocæcal angle which could give rise to gastric or duodenal ulcer can be entertained. Lymph must not only pass to the glands which guard the stomach and duodenum, but find its way *beyond* them.

Injection of the Glands along the Right Gastro-epiploic Vessels.—One of the most striking results of this investigation was achieved by an accident. In commencing an injection with indigo-carmine into the cæcal wall of a living cat, some of the dye escaped into the lower part of the abdominal cavity; as

the experiment proceeded, it was noted that the glands in the subpyloric region had become injected before the glands in the upper part of the ileocolic chain. An investigation showed that the dye had been taken up by the great omentum and delivered into this gland; further and similar experiments with both indigo-carmine and Chinese black were convincing that the subpyloric glands in cats acted as the great portal through which the peritoneal cavity in its lower part passed its effluent.

Investigations in living man on somewhat similar lines presented great

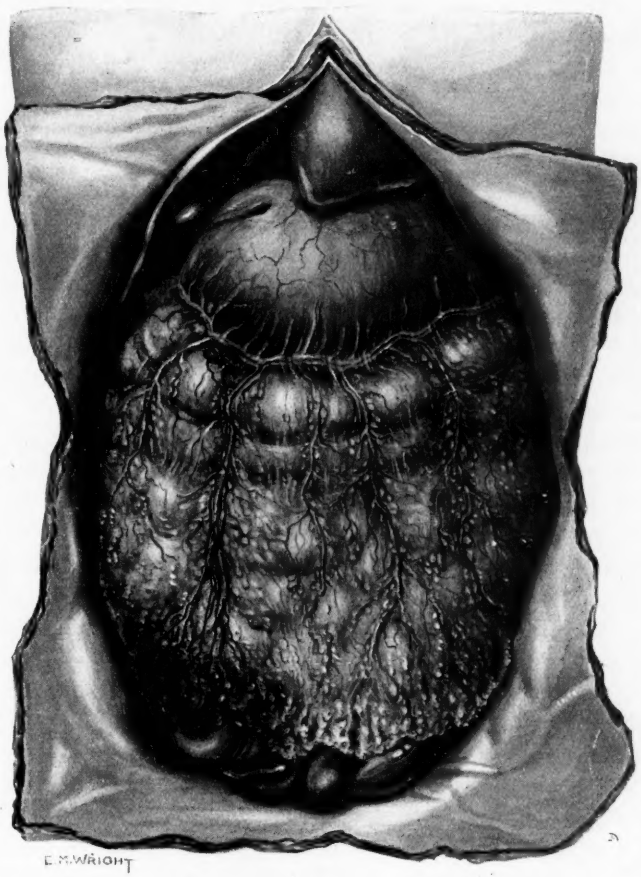


FIG. 11.—Showing flow of dye through omental lymph vessels to glands.

difficulties. It was seen early that something more than contact of the indigo-carmine with the free margin of the great omentum was necessary; it was necessary to combine it with massage. The time taken for the dye to reach the right gastro-epiploic glands was found to vary very widely; it never arrived there under one hour, and much more often failed to travel more than an inch or two in this space of time. Clearly the attempt could only be made when a major operation was in process—gastric cases could not be utilized at all. Nearly all the experiments were therefore made in cases of gall-stone

disease. In the performance of choledochotomy for common-duct stone the left forearm of the operator, and to a lesser extent the right, come to lie over and exert an intermitting pressure over the lower abdomen. It was in this class of case that the whole of the successful results were obtained. A large piece of gauze was soaked in indigo-carmine (1–60), and fixed by clips to the lower free border of the great omentum; the omentum with the attached gauze was then returned to its place in the abdomen and the operation continued. After an average time of from one to one and a half hours, streaks of the dye could be seen slowly rising along the course of the main blood-vessels of the omentum, crossing the transverse colon, and emptying into the right gastro-epiploic glands (*Fig.* 11). Out of twenty-one attempts made in suitable cases, only four were successful. With a wider distribution of the dye, two groups of lymph vessels were clearly seen, converging one to the right side of the greater curvature of the stomach and one to the left, the former filling the more rapidly and being the more clearly marked. Despite many efforts, the injection was never seen to pass through the right gastro-epiploic group on to the wall of the stomach.

The great omentum, therefore, being the great scavenger of the abdominal cavity—Moynihan calls it the great drain-pipe—is able to pick up, probably through open lymphatic terminal spaces, morbid material, and to convey it upwards to the greater curvature of the stomach, where it passes chiefly into the group of glands towards the pylorus, and in a smaller measure into those glands near the hilum of the spleen.

In connection with this work, Wilkie's[13] monograph is most interesting. He says: "The extraordinary faculty possessed by the omentum for plastic adhesion to any zone of peritoneal irritation, and thus for walling off and localizing inflammatory processes, is so constantly evident to the surgeon and to the pathologist, that they have almost come to regard the great omentum as a purely beneficent agent in abdominal pathology. Nothwithstanding its peculiar capacity for rapid reaction and repair, it would, indeed, be strange if the omentum could take part in so much inflammatory trouble and yet come out scatheless. That a chronic inflammation of the omentum may remain after the primary seat of infection has healed has been repeatedly demonstrated, but that the omentum may form the channel by which the pathological process may be carried to other organs is a fact that has hitherto received but scant recognition. The intimate anatomical relations of the omentum with the stomach and the first part of the duodenum, especially in regard to their vascular supply, suggest the possibility of morbid conditions in the former spreading or being conveyed to the latter through their vascular connections."

Wilkie then suggests that the omentum being infected by its presence in the region of the appendix, sometimes by its actual adhesion, becomes inflamed and its vessels thrombosed; he further suggests that emboli pass from these vessels to the stomach and duodenum and give rise to ulceration; he also considers that gastric erosions—by some authors confused with ulcers—thought to be the cause of hæmatemesis, and seen frequently in the absence of the real and visible ulcer, may be due to a similar pathological process, possibly to a spreading lymphangitis in the great omentum. The experiments described would certainly add weight to Wilkie's views.

POSSIBLE BEARING OF THE LYMPH FLOW ON THE CAUSE OF DUODENAL AND GASTRIC ULCER.

In considering what effect the investigations described may have on the causes of duodenal and gastric ulcer and of other forms of dyspepsia, obviously it must first be granted that the right iliac fossa is capable of producing either a toxin or an infective agent. Lane's views on this question are too well known to need any repetition. It is not within the scope of this inquiry to fix on the exact origin of the trouble; it may be the appendix, it may be the cæcum or the small intestine. There can be no doubt the appendix is the most likely of the three.

In order that the morbid effluent may reach the duodenum it should certainly pass along the lymphatic tract indicated by the experiments described. Assuming lymph to flow as described by those who have worked most on the anatomy of the lymphatic system, it is clear that, in the absence of retrograde or aberrant flow, it can never reach the duodenum. The experiments on apparently normal young post-mortem subjects—i.e., normal in regard to this tract—would appear to show that there is an easy way up to the duodenal border, and that apart from retrograde or aberrant flow. It would seem that the large groups of glands lying on or near the head of the pancreas, with the group on the superior mesenteric trunk, act normally as one big receiving station, with extensive backward and forward communications within the limits of the station: something analogous to the circle of Willis at the base of the brain, allowing lymph, hindered in its flow in one direction, to pass in another.

Let us assume that we start with perfectly healthy lymph vessels and glands. For some reason or other there comes pouring into the mesenteric group of glands a stream of infected lymph; the result is bound to be a lymphangitis and a lymphadenitis, spreading upwards from node to node with a gradually decreasing effect as it nears the last line of gland sentinels before reaching the blood-stream; assume the flow still goes on over a period of months or years. It is not too much to suggest that chronic obstructive lymphangitis and lymphadenitis develop, most markedly nearest the origin of the infection, but gradually involving the whole lymph-shed. The result of this change would be obstruction in the path of the normal flow. Gradually the whole mechanism is thrown out of gear, and we have everything present to give rise to aberrant and retrograde lymph flow. Now the infected lymph, failing to make its usual exit, seeks new avenues of escape in all directions; misses whole groups of glands which under ordinary conditions would check and filter it; and ebbs and flows to and fro until the glands around the superior mesenteric artery are reached and partly obstructed. Deprived of easy access through them to the lumbar glands, part of the lymph flows onwards over the head of the pancreas and enters the glands on the concavity of the duodenum, and in process of time bathes even the duodenal wall itself, escaping finally to the cœliac glands by normal or possibly aberrant paths, and so reaches the receptaculum chyli.

Is it not a reasonable suggestion that similar infective lymph may pass along the great omentum, and—through a similar process of obstruction in the right gastro-epiploic glands—reach the stomach? It is certainly a fair supposition in those cases where actual adhesion of omentum to the morbid

focus in the right iliac fossa has occurred; but what of those cases where there is no direct union to the omentum? What is it that takes place in an attack of acute appendicitis? There is poured out a great quantity of fluid, at first of bactericidal potency, but quickly becoming contaminated. The results of the subsequent inflammation are not confined to the appendix itself, but are frequently many inches away from it, as is shown by bands and adhesions in the neighbourhood. If this be true of acute appendicitis, may it not be true to a lesser extent of the subacute or even chronic variety?

One would venture, then, to suggest that the infective fluid is actually poured out into the general peritoneal cavity and carried in part up the great omentum to the stomach, and, as Wilkie suggests, also to the first portion of the duodenum. Is it not also possible that any infection in the abdominal cavity, the result of which is the exudation of contaminated fluid, may produce an exactly similar condition—an ascending lymphangitis in the great omentum with the same clinical and pathological results?

Following then the presence of infected lymph along the greater curvature, lasting for months or more, a state of chronic lymphangitis and lymphadenitis develops, with resulting lymph stasis, possibly an incompetency of valves, aberrant and retrograde lymph movement, and (as has been said) the stomach becomes laved by an infected fluid.

It has not been forgotten that the right gastro-epiploic glands are frequently found enlarged as a result of carcinoma of the stomach, and also as a result of simple ulcer; nor has it been lost sight of that one of the afferent paths of these glands must be gastric, the other being naturally omental; it is not pretended that gastric erosions alone (apart from ulcer) may not cause a lymphadenitis, both on the lesser and, to a smaller extent, on the greater curvature. It is unusual, however, to find the groups on both curvatures enlarged together, except in the presence of visible and palpable gastric disease. One can well imagine a small gastric lesion, or a series of small gastric lesions (erosions), set up by a flow of infective lymph, arriving via the duodenal lymphatic vessels, giving rise to enlargement of the right gastro-epiploic group of glands. When there are two groups of right gastro-epiploic glands, a small upper and a larger and more numerous lower, except in the presence of obvious ulcer, it is almost always the lower group, below the vessels, which is enlarged, i.e., the one furthest from the pylorus.

It is admitted that to accept all this without further proof needs considerable credulity and some courage; but even assuming it be accepted provisionally and for the moment, gastric and duodenal ulcer, and possibly gall-stones, are not of necessity due to it.

1. **Duodenal Ulcer.**—The presence of infected lymph in the walls of the duodenum would give rise to different conditions, depending upon the acuity of the infective agent—an acute dose of a severe infection might give rise to a catarrhal jaundice, a chronic one to a 'duodenal' dyspepsia; a slow infection of a milder type would give rise to inflammation and erosion of lymph follicles in the duodenal wall. This erosion would be likely to be countered by bile and pancreatic juice, since they are alkaline, and so would not give rise to ulceration below the bile papilla; and the erosions would speedily heal up. That portion of the duodenum most likely to suffer would be above the

bile papilla, where the contents are acid, and would be in the area least supplied with blood, i.e., Mayo's anæmic spot on the anterior wall of the first part of the duodenum towards the upper or antimesenteric border, and this, of course, is the usual place for a duodenal ulcer. It is in this area also that most of the masses of lymphoid tissue are normally present.

2. **Gastric Ulcer.**—Assuming the pyloric half or third of the stomach to be bathed from time to time in a wave of infected lymph, how can this produce the usual type of gastric ulcer in the usual situation? The early effect on the stomach would be: congestion (pyloric blush) with hypersecretion, hyperacidity, and spasm—the conditions found to be present in appendix dyspepsia (Moynihan[1] and Paterson[2]). Later on, multiple gastric erosions would occur, perhaps giving rise to hæmatemesis (the common type of so-called 'gastric ulcer'). Bolton[14] says: "Perhaps the commonest cause of hæmorrhage into the mucous membrane is bacterial infection—either by direct action of the bacterial poison on the capillary wall, or by giving rise to vascular occlusion and hæmorrhagic infarction; in the latter case necrosis of tissue is always present, in the former it may not be". Letulle[15] certainly produced gastric ulcer by inoculation of the general peritoneal cavity by the *Staphylococcus pyogenes aureus*, probably via the great omentum. Relics of a bacterial infection are to be found in the crater of many gastric ulcers (Bolton, Balfour). Acute ulcers are known to occur in many infective diseases, e.g., puerperal fever, peritonitis, pleurisy, tuberculosis (Jaksch, 1844). All or most of the erosions would heal during a temporary lull in the flow of infection, but one or more might remain to become a true gastric ulcer.

Why, then, should the effects of the poisoned lymph be permanent only on the lesser curvature and near the pylorus? (1) It is in this place that lymph follicles are twice as numerous as, and are bigger than, in any other portion of the stomach (Bolton); (2) The lesser curvature is the chief channel for the transmission of the contents in a stomach which is not full (Lewis); (3) The lesser curvature is the antimesenteric border, the ulceration thus coming into line with the usual antimesenteric ulceration seen in other parts of the gastro-intestinal canal—possibly due to a decreased blood-supply.

If, then, ulcer of stomach or duodenum be due to infection received from the appendix, the elimination of the offending organ might be expected to cure the ulcer. There is no doubt appendicectomy frequently cures an indigestion which cannot be differentiated from duodenal or gastric ulcer. Bolton and Stewart[16] have shown that there are frequently many scars of healed ulcers present post mortem, and there can be no doubt that most cases of chronic indigestion are accompanied by curable ulceration. Assuming, however, that a really well-marked ulcer is developed, the presence of free HCl, and the persistence of trauma and mobility, may cause it to be intractable even in the event of the original cause being cut off. The constant desire of the ulcer to heal is seen in the presence of massive fibrous barriers which frequently surround it, as well as in the typical periods of symptomatic quiescence.

If, then, there are grounds for believing that duodenal and gastric ulcers can arise in this way, are there not almost equal grounds for attributing the *B. coli* infection of the gall-bladder to a similar phenomenon?

Cases showing actual and obvious pathological extension by a direct lymphatic connection from the ileocæcal angle to the region of the pyloric end of the stomach are not far to seek. The pigmented-gland case has already been described. Moynihan recently, whilst engaged in an abdominal operation, found a mass of tuberculous glands in the ileocæcal angle, a complete chain up to the third part of the duodenum, and in addition a chain running along the common bile-duct; so far had the process of calcification proceeded in the latter group that he was momentarily led to the belief that he had to deal with stones in the common bile-duct. Carlton Oldfield had a similar case. Gordon Taylor quite recently related a remarkable case in which the patient, having previously had a gastro-enterostomy performed, developed the signs and symptoms of perforated gastrojejunal ulcer in the line of the anastomosis. Operation revealed the perforation with a generalized peritonitis. As part of a routine examination the appendix was examined, and found to be in the stage of acute gangrenous inflammation. This case would suggest infection of the posterior wall of the stomach from a flow of highly-infected lymph. Moynihan has related several cases of the co-existence of acute appendicitis and acute cholecystitis occurring in his own practice.

The investigation described would lead one to suggest that a new anatomy of the living as well as a new pathology of the living, or the two combined, might be deemed at any rate worthy of serious consideration.

The author desires to acknowledge the kindly interest and assistance given him in the preparation of this paper by Dr. R. A. Veale, Mr. J. T. Blackburn, Professor M. J. Stewart, and the Medical and Pathological Departments of the University of Leeds. The X-ray photographs were taken and very kindly lent by Dr. Leo. A. Rowden.

REFERENCES.

[1] Moynihan, Sir B. G. A., "Remarks on Appendicular Dyspepsia", *Brit. Med. Jour.*, 1910, Jan. 29.
[2] Paterson, H. J., "Appendicular Gastralgia, or the Appendix as a Cause of Gastric Symptoms", *Proc. Roy. Soc. Med.*, 1910, Part III, Surg. Sect., 187.
[3] Moynihan, Sir B. G. A., *Duodenal Ulcer*, 2nd ed., 1911.
[4] Cuneo and Delamere, "The Lymphatics of the Stomach", *Jour. de l'Anat. et de la Physiol. Normales et Path. de l'Homme et des Animaux*, 1900, 393.
[5] Moynihan, Sir B. G. A., *Abdominal Operations*, 1914, i, 291.
[6] Jamieson and Dobson, "Lectures on the Lymphatic System of the Stomach", *Lancet*, 1907, April 20.
[7] Poirier and Charpy, *Traité d'Anatomie Humaine*, 1909, ii.
[8] Stiles, Sir H. J., "Contributions to the Surgical Anatomy of the Breast and Axillary Lymphatic Glands", *Edin. Med. Jour.*, 1892, July, 26.
[9] McVay, J. R., "The Lymph Node in Carcinoma of Rectum", *Ann. of Surg.*, 1922, Dec.
[10] Wiener, Joseph, "Ileocæcal Tuberculosis", *Ibid.*, 1914, lix, 698.
[11] Broders, A. C., "Tuberculosis of the Stomach, with report of a case of Multiple Tuberculous Abscess", *Surg. Gynecol. and Obst.*, 1917, xxv, 490.
[12] Most, A., *Chirurgie der Lymphgefaesse und der Lymphdruesen*, 1917.
[13] Wilkie, D. P. D., "Retrograde Venous Embolism as a Cause of Acute Gastric and Duodenal Ulcer", *Edin. Med. Jour.*, 1911, May.
[14] Bolton, Chas., *Ulcer of Stomach*, 1913.
[15] Letulle, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris*, 1888, Aug. 10, 330.
[16] Stewart, M. J., "The Healing of Gastric Ulcer", *Brit. Med. Jour.*, 1922, Dec. 16.

ON UNILATERAL FUSED KIDNEY AND ALLIED RENAL MALFORMATIONS.

By M. J. STEWART and S. D. LODGE, Leeds.

(From the Department of Pathology and Bacteriology, Leeds University.)

Among the many developmental anomalies to which the urinary apparatus is subject, that of fusion of the kidneys occupies an important place. It is met with in two forms, of which one, the horseshoe kidney, is common, while the other, the 'concrescent' kidney of Gérard, is rare.

In the horseshoe group the kidneys occupy more or less their normal position in the body, but are united across the middle line by a bridge of renal or, less commonly, fibrous tissue. In the great majority of cases it is the lower poles which are so joined; exceptionally the junction is at the upper poles. Of 90 cases collected by Gérard (1905),[6] 83 belonged to the former class, and only 7 to the latter. Morris (1901),[13] in a search through the records of four London hospitals, found 19 cases of horseshoe kidney in 18,244 post-mortems. In a series of 6500 post-mortem examinations at the Leeds General Infirmary there were 14 cases of horseshoe kidney, all showing fusion below.

The 'concrescent' group of Gérard consists of cases where, as a rule, renal fusion is much more extensive, and where there is more or less asymmetry of renal tissue, with absence of the horseshoe form. It is further subdivided into those cases where the fused mass is, in part at least, prevertebral, and those where it is definitely and completely unilateral. We have ventured to add a third variety, the pelvic fused kidney.

This classification, based on Gérard, may be shown thus :—

Table I.—Fusion of the Kidneys.

1. *The horseshoe kidney* :—	(*a*) Fusion at upper poles.
	(*b*) Fusion at lower poles.
2. *The 'concrescent' kidney* :—	(*a*) The prevertebral fused kidney.
	(*b*) The unilateral fused kidney.
	(*c*) The pelvic fused kidney.

The classification here suggested is an important one from the clinical standpoint, and is to be preferred to the purely morphological subdivision of class 2 (above) into sigmoid kidney, where there is more or less end-to-end anastomosis, and disc-shaped kidney, where the amalgamation is much more complete (Morris, 1901,[13] Newman, 1898[14]). As used by Newman, however, the terms practically correspond: sigmoid kidney with unilateral fused kidney, and disc-shaped kidney with prevertebral fused kidney. Disc-shaped kidney, on the other hand, is sometimes unilateral.*

* The terms 'unsymmetrical kidney' and 'solitary kidney', meaning respectively congenital absence of one kidney and fused kidney, formerly much in use, seem to us merely misleading. Fused kidney, for example, may be 'unsymmetrical', as in the case described in detail in this paper.

Gérard collected 8 cases of prevertebral fused kidney from the literature, and 14 of the unilateral type. He also refers to 4 cases of pelvic fused kidney, those of Cruveilhier, Dubor, Carrien, and De Rouville. A fifth instance, from the Museum at Guy's Hospital, is given by Wilks and Moxon (1889).[19] The only other case of this kind of which we have found a record is one mentioned by Duckworth (1869).[5] He says, ". . . I have found the notes of one (case) where there was only a right kidney present. It was an intrapelvic organ in this instance, however. The specimen is in the Pennsylvania Hospital Pathological Museum. It has two ureters, which enter the bladder at the usual place, and two renal arteries. The general outline of the organ is described in the catalogue as circular". We have been unable to find any published account of this case beyond Duckworth's brief reference, and while the description of the ureters is ambiguous, it seems at least highly probable that this is an example of pelvic fused kidney.

In our own series of 6500 autopsies we have met with but one example of unilateral fused kidney and none of the other two varieties. Morris found "one fused kidney other than horseshoe-shaped" in 15,908 autopsies.

Table II.—Incidence of Congenital Renal Abnormalities in a Consecutive Series of 6500 Autopsies.

Type of Abnormality		No. of Cases	Incidence
1. Horseshoe kidney		14	0·21 per cent
2. Unilateral fused kidney ..		1	
3. Congenital absence of kidney—			
Right absent ..	4	16	0·24 ,,
Left absent ..	12*		
4. Pelvic kidney		3*	0·04 ,,

* One case is common to these two groups.

By contrast with these anomalies, the *congenital absence* of one kidney appears to be fairly common. Gérard collected no fewer than 279 cases from the literature, and in our series there were 16 cases. Morris, on the other hand, found but 6 cases in 15,904 autopsies from Guy's Hospital, the Middlesex Hospital, St. Bartholomew's Hospital, and the Hospital for Sick Children, Great Ormond Street. Apparently 3 of these were regarded as cases of congenital absence, and 3 as examples of extreme atrophy or extreme want of development. According to these figures, absence of a kidney is met with once in every 2650 post-mortems, whereas in our series the incidence is one in every 400. From the practical standpoint it makes little difference whether the absence of the kidney is due to purely developmental defect or to extreme atrophy during fœtal life. Nevertheless, in our series of 16 cases we feel confident that at least the great majority are examples of genuine congenital (developmental) defect. In the first place, careful naked-eye examination failed to reveal even the tiniest shred or relic of renal tissue

on one side of the body. In the second, it is specifically noted in 5 cases that the ureter on that side was also lacking, and that in 3 of these there was no sign of an orifice or plica ureterica on the corresponding side of the bladder. In the third place, 5 cases (4 of them other than those with absence of the ureter) showed other important developmental abnormalities, viz., absence of homolateral adrenal (1 case), bicornuate uterus (1 case), absence of homolateral testis (1 case), and imperfectly descended testis (2 cases).

1. **Horseshoe Kidney.**—All the cases of horseshoe kidney were of the usual type: the organs fused at their lower poles by a thick band of renal tissue. The ureters invariably passed down in front of the fused organ. In the cases in which there was any displacement, the kidneys merely occupied a slightly lower position than usual in the body, or approximated more nearly to the middle line. No other developmental abnormalities were noticed in these cases.

The age of these patients is of interest, as showing that the possession of a horseshoe kidney is no bar to long life. The average age at death is 47 years. One patient died at 82, following an operation for strangulated inguinal hernia. Two patients were aged 60 to 70, five 50 to 60, two 30 to 50, and two 20 to 30, while the youngest was 14. This compares very favourably with the general age-distribution of the 6500 autopsies. In only one case was there gross kidney disease—chronic nephritis in a woman of 27.

2. **Unilateral Fused Kidney.**—This case is referred to in detail later; it is sufficient to say here that the condition was unaccompanied by any other congenital malformation, nor was it related to the death of the patient.

3. **Congenital Absence of one Kidney.**—As shown in *Table II*, the incidence of this defect in the present series is much the same as that of horseshoe kidney. The much greater frequency of left-sided defect (3–1) is not quite in keeping with Gérard's larger series of cases. There, in the 232 cases in which the side is mentioned, the left kidney was absent in 136, the right in 96, a ratio of $3\frac{1}{2}$ to $2\frac{1}{2}$. Sex-distribution is also unequal. In our series there were 14 males and 2 females. In Gérard's collected cases there were 122 males and 93 females. This latter discrepancy may be accounted for by the fact that more males than females come to post-mortem.

The weight of the single kidney is usually above normal, but rarely reaches that of two healthy kidneys. In our cases the usual description is that the organ was 'slightly enlarged' or 'moderately enlarged'. Of six adult kidneys weighed, the largest was 10 oz., and was the seat of subacute nephritis; three were between 7 and $7\frac{1}{2}$ oz., and two were $3\frac{3}{4}$ and $2\frac{1}{2}$ oz. respectively. The last was the seat of advanced chronic interstitial nephritis.

As compared with the cases of horseshoe kidney, the incidence of renal disease is high in this group. In 6 out of the 16 cases, the death of the patient was directly attributable to disease of the solitary kidney. There were 3 cases of subacute and chronic nephritis, 1 of calculous pyelonephritis, 1 of tuberculosis, and 1 of ascending pyelonephritis following cystitis. This, of course, is a much higher incidence of renal disease than in the average run of post-mortems. In the remaining 10 cases there was no evidence to show that absence of one kidney had any connection whatever with the death of the patients. Two died in infancy and 3 in childhood, but of the remaining 10

no fewer than 6 were 55 years of age or over. The oldest patient was 81, and he died from a fractured skull.

The average age at death in this group is 36·5 years, but if the two infants, who died of rickets and hydrocephalus respectively, be deducted, the figure is 42·5, as compared with 47 years in cases of horseshoe kidney.

Statistically these figures are too few to be reliable, but they tend to show that the possessors of a single kidney are shorter-lived and more prone to renal disease than those who have either a pair of kidneys or a horseshoe organ. Unfortunately this aspect of the subject is not touched on by Gérard.

The frequency of other congenital malformations, especially of the genital tract, in cases of congenital absence of the kidney, is insisted on by all writers on this subject (Gérard, etc.). In one of the two female cases in our series there was a bicornuate uterus. In 3 of the 14 male cases there were testicular abnormalities on the same side as that from which the kidney was missing. In one of them the testis was absent, in another it was in the inguinal canal, and in the third it lay in the abdomen at the outer edge of the psoas muscle. It is fairly certain, moreover, that in some of our cases the genital tract was not carefully examined, and in this way defects may have been missed. Of 8 cases where the adrenals are specifically referred to in the post-mortem report, in 7 these glands were normally situated ; in 1 the homolateral adrenal was missing.

4. **Pelvic Kidney.**—Two of these cases are of the usual type. One kidney (the right), malformed, was situated in the pelvis, and obtained its blood-supply by a short renal artery springing from the abdominal aorta at or near its bifurcation. The patients were a male of 53 and a female of 66, and in neither was the renal condition in any way related to the cause of death. The third case is a very unusual one, and worthy of fuller notice. Here the left kidney was congenitally absent, and the solitary right kidney was situated in the pelvis. It was connected to the bladder by a short, kinked ureter only 3 inches in length, and obtained its blood-supply by a single large artery given off at the bifurcation of the aorta. The left ureter, like the kidney, was non-existent. The patient, a man of 55, died of an acute ascending pyelonephritis following cystitis of unexplained origin.

This condition must be very rare. The only case of the kind mentioned by Gérard is that of Polk (1883),[15] which is of extraordinary interest in that the solitary (ectopic) kidney was removed surgically. The patient, a girl of 19, had a movable and painful tumour in the left iliac fossa. It was oval, with its long axis directed downwards and inwards. The genital organs were rudimentary, the uterus and vagina being absent. The tumour was thought to be kidney, and as it was easily accessible and seemed to be the seat of the violent pain of which the patient complained at each menstrual period, it was removed. The organ, after excision, appeared healthy and weighed 198 grm. Death ensued eleven days later, and at autopsy it was found that the right kidney and ureter were completely absent.

In addition to the 3 cases of pelvic kidney just cited, there is, in our series, 1 case in which the fixed and displaced organ lay just above the brim of the pelvis. It has not been included in the statistical table.

Pelvic kidney may occupy either a median or a lateral position, and it is invariably much deformed. Where the kidney sits on the pelvic brim it usually lies over one or other sacro-iliac synchondrosis.

REPORT OF A CASE OF UNILATERAL FUSED KIDNEY.

Except for the fact of its obvious rarity, the unilateral fused kidney is a condition of great clinical interest and importance, and, inasmuch as the ureters in these cases invariably open normally into the bladder, the true state of affairs, even after careful cystoscopy, may easily be missed. The following example illustrates very well the possible value of pyelography in such cases.

The patient was a man of 56 years, who died of acute generalized peritonitis following left inguinal colostomy for annular carcinoma of the sigmoid flexure. There were early metastatic deposits of growth both in the liver and in the abdominal (pre - aortic) and mediastinal lymph glands.

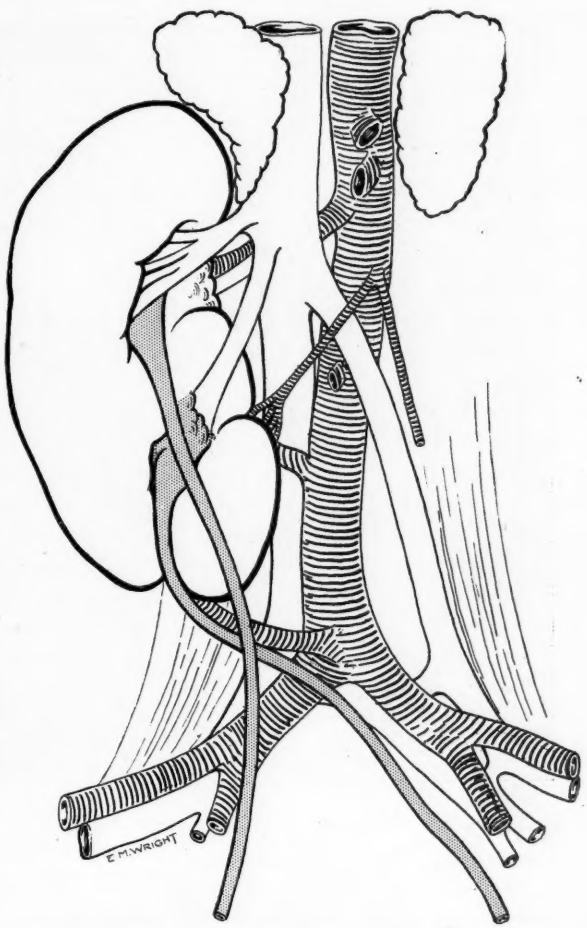


FIG. 12.—Drawing of the specimen of unilateral fused kidney, with its vascular connections.

Kidneys.—There is no renal tissue on the left side of the body, although the left adrenal is normally situated. The right kidney (*Fig.* 12) is apparently enlarged, measuring 16·5 × 7·5 × 4 cm. in its three principal diameters. Attached to and closely incorporated with the inner aspect of its lower half is an accessory kidney, measuring 9 cm. in length, and about 3·5 cm. in width. This distinctly demarcated mass would seem to represent the left kidney, and, even so, the two organs appear to be fused over a wide area. Each has a separate hilum, pelvis, and ureter. On its posterior aspect the conjoined organ presents a very much flattened and altogether more uniform surface, the only sign of duplication being the entry of a large artery into the middle of the renal mass. The exact relation of the two organs to one another is accurately brought out by radiographic examination, after injection of both ureters with collargol solution (*Fig.* 13). It is now seen

that the external appearances are deceptive, and that practically the whole of the lower half of the renal mass represents the left kidney. The complete independence of the pelvis and calices of each organ is also shown. The total weight of the fused organs in the fresh state is 252 grm., i.e., within normal limits for the kidneys taken together. The volume after formalin fixation is 202 c.c. The adrenals are normally situated.

Pelves and Ureters (*Fig.* 12).—The hilum of the normally situated kidney is directed forwards and inwards, that of the transposed organ almost directly forwards. That the upper half of the renal mass is the normally situated right kidney, and the lower half the transposed left kidney, is confirmed by the position and course of the ureters. The right ureter springs from the upper hilum, roughly at the level of the 1st lumbar vertebra, this pelvis having one vein anterior to it and an artery and vein behind. The ureter runs downwards and slightly inwards over the fused organ, crosses the other ureter near the upper end of the right common iliac artery, and after a normal pelvic course opens into the right side of the bladder. Its length is about 28 cm.

FIG. 13.—Radiogram of the specimen after removal from the body. The two pelves and their calices have been injected with collargol solution.

The other ureter (the left) springs from the lower hilum, and at first runs almost directly downwards in a deep groove in the anterior surface of the kidney. It then passes downwards and inwards across the right psoas magnus muscle, passes beneath the right ureter, and crosses the middle line just below the bifurcation of the aorta. Thereafter it pursues a normal pelvic course, to enter the left side of the bladder in its proper position. Its length is about 26 cm.

Arteries (*Fig.* 12).—The right renal artery springs from the aorta immediately below the level of the superior mesenteric. It passes to the right behind the inferior vena cava and renal vein, and after a course of 6·5 cm. enters the hilum of the right kidney proper behind the pelvis. A second, much smaller artery arises from the left side of the aorta a short distance below the first, and passes downwards and to the right, crossing in front of the

aorta and inferior vena cava. After a course of some 7 cm. it divides into four branches, which enter the inner border of the original left kidney. A third artery of considerable size springs from the right side of the aorta 4·5 cm. above the bifurcation, and runs upwards, backwards, and outwards for a distance of about 8 cm., entering the posterior surface of the renal mass 6 cm. above the lower pole and midway between the outer and inner borders. A fourth fairly large artery springs from the front of the bifurcation of the aorta and runs upwards, backwards, and to the right for a distance of 7 cm., to enter the lower hilum behind the pelvis. Thus, of these various arteries, it would appear that the first supplies the right kidney; the second, third, and fourth the left. It is possible, however, that the third, a large vessel, is distributed to both. No injection experiments were made on the blood-vessels.

Veins.—As shown in *Fig.* 12, the left external iliac vein, after a communication with the internal, passes up on the left of the aorta, which it crosses to join the inferior vena cava at the level of the 2nd lumbar vertebra. There are three renal veins in all, one from the lower hilum and two from the upper. All three join the inferior vena cava about the level of the 12th dorsal vertebra, and just above the point of entrance of the left external iliac.

Bladder.—The orifices of the ureters and plicæ uretericæ appear normal.

Remarks on Unilateral Fused Kidney.

Gérard (1905)[6] collected 14 cases of unilateral fused kidney from the literature. We have found records of 13 others, making, with the one here reported, a total of 28.

This list is the result of an extensive search through the literature, but is probably incomplete. Papers by Hortolès (1882)[10] and Thoreus (1870)[18], for example, which we have not been able to obtain, probably relate to this subject.

Cases of unilateral fused kidney are all much alike, at least in their salient characteristics. They differ from one another chiefly in respect of their vascular connections, and to some extent in the size and shape of the fused mass. The amount of renal tissue present is usually rather less than that of two normal kidneys. In the case reported by Kelly (1868),[11] however, it is stated that the fused organs weighed 93 oz. No explanation of this extraordinary finding is given. Some idea of the variations in shape is afforded by the drawings illustrating this paper (*Figs.* 12, and 14–19).

In the great majority of cases the displaced organ lies below the normally situated one, its upper pole fused with the lower pole of the latter. There would appear to be no rotation in the process of transposition, inasmuch as the hilum remains directed to the same side as would have been the case had the organ remained in its normal position. This is well seen in Broesike's case (1884),[2] and less definitely in the present instance. The true nature of the ectopia is clearly shown by the course of the ureters. Invariably these arise from separate and clearly-defined pelves lying one above the other. The ureter from the upper pelvis passes down to enter the same side of the bladder; that from the lower crosses the middle line and enters the bladder on the opposite side.

The cases reported by Dickinson (1895)[4] and by Kidd (1910)[12] are exceptional in that it is the upper half of the organ which is ectopic.

Table III.—List of Published Cases of Unilateral Fused Kidney.

Reported by	No. of Cases
Gérard, 1905[6]	14
Hunter, 1793; Horne, 1793; Sandifort, 1793; Chassaignac (2), 1832, 1840; Reed, 1845; Stoicesko, 1877; Stocquart, 1880; Poulalion, 1890; Powell, 1883; Tesson, 1895; Chambrelent, 1895; Cathelin, 1898; Tanton, 1901.	
Hillier, 1864[9]	1
Kelly, 1868[11]	1
Coupland, 1877[3]	1
Greenfield, 1877[7]	1
Broesike, 1884[2]	1
Birmingham, 1890[1]	1
Dickinson, 1895[4]	1
Sutherland and Edington, 1898[16]	2
„ „ 1900[17]	1
Kidd, 1910[12]	1
Gruner and Fraser, 1911[8]	2
Stewart and Lodge	1
Total	28

Here the upper ureter crosses to the other side and enters the bladder in its proper place, i.e., on the side from which the kidney is absent.

Fig. 14.
Coupland's case.

In contradistinction to cases of congenital absence of one kidney, where multiple defects are common, unilateral fused kidney is rarely associated with other defects of the genito-urinary tract. Similarly, in all reported cases, the suprarenal glands have occupied practically their normal position.

There is not sufficient evidence to show that fused kidney is more common on one side than the other. Gérard found that the fused organ occurred most frequently on the right side, but in the 14 cases which we have collected it was more common on the left, the ratio being 9 to 5.

The vascular arrangements are anomalous. Usually three or four arteries pass to the fused organ, of which two supply the upper and two the lower half. One or both of the vessels for the upper half spring direct from the aorta, in or below the normal position. The vessels for the lower half are more irregular. They may arise from the lower part of the aorta, notably at the level of the bifurcation, or they may spring from the common

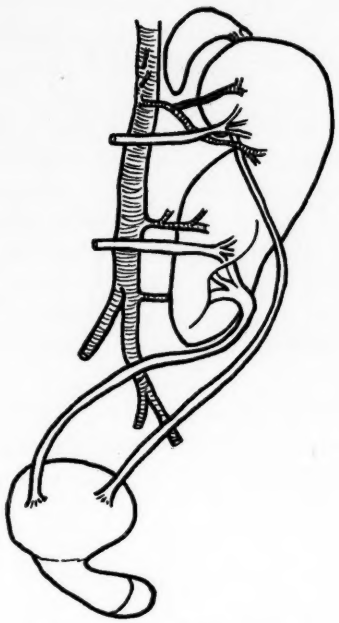


FIG. 15.—Broesike's case.

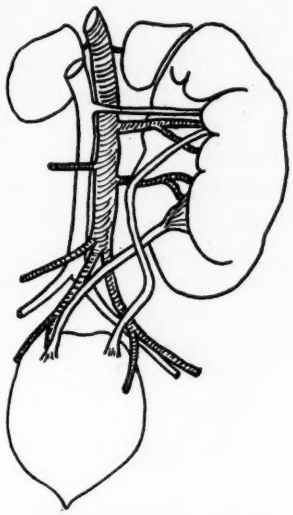


FIG. 16.—Sutherland and Edington's *Case* 1.

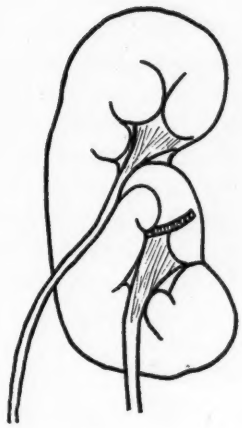


FIG. 17.
Sutherland and Edington's *Case* 2.

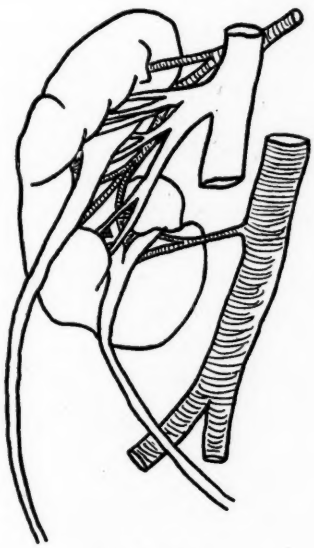


FIG. 18.
Sutherland and Edington's *Case* 3.

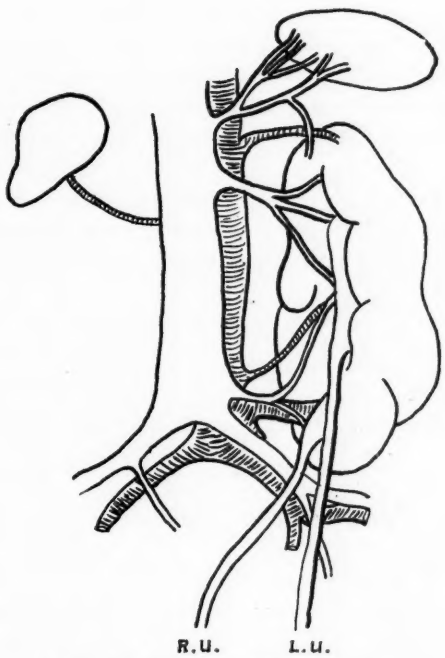


FIG. 19.—Gruner and Fraser's case.

FIGS. 14 TO 19.—DRAWINGS FROM A NUMBER OF PUBLISHED CASES OF UNILATERAL FUSED KIDNEY.

iliac artery of the opposite side (Birmingham, 1890).[1] In Greenfield's (1877)[7] case there was but one renal artery, which presently bifurcated to supply the two portions. In Sutherland and Edington's (1900)[17] third case there was one artery to each hilum.

We desire to emphasize the clinical importance of cases of this kind. It requires but little thought to appreciate the possible consequences should tuberculous, calculous, or neoplastic disease of the normally situated organ call for surgical intervention. Cystoscopic examination or even urethral catheterization would apparently reveal a normally functioning kidney on the side away from the lesion, and it might well be at a late stage of nephrectomy that the surgeon would discover he was dealing with a developmental abnormality of some kind. Even then he might well assume the presence of nothing more unusual than a double ureter.

Radiological examination in such a case would, of course, be of the greatest possible service, since it would show that the normal renal shadow was lacking on one side, while the passage of opaque bougies, or (preferably) pyelography, would completely establish the diagnosis.

SUMMARY.

The congenital renal abnormalities found in a consecutive series of 6500 post-mortem examinations are described. The list includes 14 cases of horseshoe kidney, 1 of unilateral fused kidney, 16 of congenital absence of one kidney, and 3 of 'pelvic' kidney. The cases in which horseshoe kidney was found are compared with those in which one organ was congenitally absent, with respect to (*a*) the incidence of renal disease, and (*b*) the age at death.

The case of unilateral fused kidney is described in detail, and the published cases of this condition are collected and reviewed.

We beg to express our indebtedness to Dr. Leo. A. Rowden for the radiogram of the injected specimen of unilateral fused kidney, and to several writers on the subject of renal abnormalities for permission to copy their drawings.

REFERENCES.

[1] Birmingham, A., "Single Unilateral (Sigmoid) Kidney", *Irish Jour. Med. Sci.*, 1890, xc, 47.

[2] Broesike, G., "Ein Fall von congenitaler S-förmiger Verwachsung beider Nieren", *Virchow's Arch.*, 1884, xcviii, 338.

[3] Coupland, Sidney, "'Solitary' Kidney", *Trans. Pathol. Soc., Lond.*, 1877, xxviii, 159.

[4] Dickinson, W. Lee, "'Solitary' Kidney", *Ibid.*, 1895, xlvi, 80.

[5] Duckworth, Dyce, "Single Kidney, with Compensatory Enlargement", *Ibid.*, 1869, xx, 232.

[6] Gérard, G., "Les Anomalies congénitales du Rein chez l'Homme", *Jour. de l'Anat. et de la Phys. norm. et path. de l'Homme et des Animaux*, 1905, xli, 241, 411.

[7] Greenfield, W. S., "Single Kidney", *Trans. Pathol. Soc. Lond.*, 1877, xxviii, 161.

[8] Gruner, O. C., and Fraser, J. R., "A Case of Fused Kidney, with a Study of Allied Deformities of the Kidney", *Bull. Roy. Vict. Hosp., Montreal*, 1911, i, 74.

[9] Hillier, T., "Double Pneumothorax from Rupture of Air-vesicles, Textural Change and Thickening of Diaphragm. Solitary Kidney", *Trans. Pathol, Soc. Lond.*, 1864, xv, 43.

[10] HORTOLÈS, " Union intime de deux Reins simulant un Rein unique ", *Mém. et Compt-rend. Soc. d. Sci. méd. de Lyon* (1881), 1882, xxi, pt. 2, 30.
[11] KELLY, C., " Abnormal (Single Unsymmetrical) Kidney ", *Trans. Pathol. Soc. Lond.*, 1868, xix, 274.
[12] KIDD, FRANK, *Urinary Surgery*, 1910, 31. London: Longmans, Green & Co.
[13] MORRIS, HENRY, *Surgical Diseases of the Kidney and Ureter*, 1901, vol. i, 58. London: Cassell & Co.
[14] NEWMAN, DAVID, " Malformations of the Kidney and Displacements without Mobility ", *Trans. Clin. Soc. Lond.*, 1898, xxxi, 118.
[15] POLK, W. M., " Case of Extirpation of a Displaced Kidney ", *N.Y. Med. Jour.*, 1883, xxxvii, 171.
[16] SUTHERLAND, L. R., and EDINGTON, G. H., " A Series of Specimens illustrative of certain Congenital Affections of the Urinary Apparatus ", *Glasgow Med. Jour.*, 1898, xlix, 81.
[17] SUTHERLAND, L. R., and EDINGTON, G. H., " A Second Series of Specimens illustrative of certain Congenital Affections of the Urinary Apparatus ", *Glasgow Hosp. Rep.* 1900, iii, 462.
[18] THOREUS, " Rein unique par Fusion ", *Bull. Soc. anat. de Paris*, 1870, xlv, 413.
[19] WILKS, S., and MOXON, W., *Lectures on Pathological Anatomy*, 3rd ed. 1899, 511. London: Longmans, Green & Co.,

METASTATIC TUMOURS OF BONE.*

By CECIL A. JOLL, London.

The object of this paper is to show that a knowledge of bone metastases, their mode of production, the types of neoplasms which give rise to them, and their special clinical manifestations, is essential not only in the study of pathology, but also in the practice of surgery.

MODE OF ORIGIN.

Secondary tumours in bones must of necessity arise in one of three ways: (1) By direct extension of a tumour from tissues outside the bone into its substance—a method which I exclude, since it is incompatible with the definition of a metastasis as I understand it; (2) By extension through the blood-stream—either by malignant emboli conveyed through nutrient arteries, or possibly by a retrograde route through the veins; or (3) By extension through lymphatic paths—a view which is compatible with either an embolic or a permeation conception of the origin of such metastases. With the latter theory the name of Sampson Handley[1] is prominently associated.

In order to prove whether metastasis in bone occurs through the lymph stream, a knowledge of the lymphatics of the periosteum, bone, and bone-marrow is imperative. It is not sufficient to show that the deep fascial lymphatics extend as far as the periosteum; they must be traced, if possible, into the depths of the bone. Piney, [2, 3] in his recent investigations, has been able to confirm the statement of Roger and Josué,[4] that it is impossible to demonstrate lymphatics in any part of the bone-marrow. He used the technique of Dewey and Noyes,[5] and found that the injection material could be forced through the compact bone, but never into the marrow proper. It was always arrested at the endosteum. This information must cast doubt at once on the opinion held by Handley that, in carcinoma of the breast, the metastases in bones are the result of centrifugal permeation of lymphatic vessels.

The only theory remaining to explain secondary deposits in the bones is that they reach the bone-marrow by way of the blood-stream. It was von Recklinghausen[6] who first elaborated this conception—viz., that metastases in bone are the result of the arrest of malignant emboli in marrow capillaries.

In order to substantiate this claim it is necessary to demonstrate: (1) That malignant emboli circulate in the blood-stream; (2) That such emboli are able to gain access to the systemic circulation; (3) That these emboli can obtain lodgement in the marrow blood-vessels.

---

*This paper is based on a Hunterian Lecture delivered by the writer at the Royal College of Surgeons in 1923.

It may be, as Nepveu (quoted by Roger Williams[7]) surmised, that the malignant cell does not circulate as such, but in the form of minute cellular bodies, smaller than the fully developed cancer cell. This would make it easy to explain how it is possible for the metastatizing element to pass through the pulmonary capillaries. No confirmation of such minute cellules is forthcoming. On the other hand, Schmorl[8] has been able to show that portions of chorionic villi are shed into the circulation in eclampsia, and Veit[9] later demonstrated that this may occur in normal pregnancy. By analogy we should expect that malignant cells also would pass into the circulation. Goldmann[10] claims that this does occur, and that the malignant cells reach the blood-stream by entering the lumina of smaller vessels, especially the veins, via the vasa vasorum. It can be seen with the naked eye that tumours such as hypernephromata grow directly into the larger veins, so that particles are readily washed away into the right heart. It is equally easy to understand that particles of growth may be conveyed along the thoracic duct and other large lymphatic vessels, and so directly into the main veins. Leaf[11] and others have demonstrated that there are numerous communications of a less obvious character between the lymphatic and blood-streams, e.g., in the hæmolymph glands.

It is possible for malignant emboli to pass from the right side of the heart directly into the systemic circulation in those exceptional circumstances when some defect exists in the inter-auricular septum. In other cases the cells must either pass the pulmonary capillaries, or, when arrested in these capillaries, they must grow into the smaller radicles of the pulmonary veins, and again becoming free as emboli, so reach the left side of the heart. Schmidt[12] has demonstrated that such minute malignant emboli are in fact actually found in many cases in the pulmonary arterioles in the interior of thrombi, without any evidence of metastases in other parts of the body. These minute malignant pulmonary foci appear to remain latent, only exceptionally shedding emboli into the systemic circulation. Of these probably only a few survive to be arrested elsewhere in the body and produce metastases.

Von Recklinghausen[6] believed that secondary growths in bone were due to such minute malignant cell masses being held up in the capillaries of the bone-marrow. He based this belief on the absence of lymphatics and the existence of a definite endothelial-lined space enclosing the malignant cells. Erbslöh[13] carried this a step further by proving that, in addition to the malignant cells, red blood-corpuscles could also be found enclosed in the same vessel. Piney[2, 3] contrasts the well-formed blood-vessels of the fatty marrow with the innumerable thin-walled capillaries of the red marrow, and sees in the latter a clear explanation why malignant emboli are arrested in the red marrow. His contention is that with the immense widening of the blood-stream which occurs as we pass from the yellow to the red marrow, there is a corresponding diminution in the velocity of the stream, and that malignant cells tend to cling to the vessel wall and so come to rest, just as the leucocytes do under similar circumstances.

It follows from Piney's claims that all bone metastases should be found in the red marrow, and therefore a knowledge of the distribution of this, under varying conditions of health and age, is necessary. Piney[3] has shown that

while in children the red marrow fills the marrow cavity, at about puberty this begins to be replaced by fatty marrow, but that while this change is completed in the distal bones of the limbs, a considerable amount of red marrow persists in the upper ends of the humerus and femur. In the epiphyses, too, this transformation from red to fatty marrow occurs with puberty and adolescence, though small foci of red marrow may persist throughout life. He was able to prove microscopically that throughout the fatty marrow minute foci of cellular red marrow could be found. A large quantity of cellular marrow can also be found in the ribs, vertebræ, sternum, pelvis, and skull bones, and smaller amounts in the clavicle and scapula.

How, then, does the location of bone metastases accord with these findings as to the distribution of the red bone-marrow? The specimens illustrated in this paper provide a general confirmation of the contention that these two are interdependent; but there are certain exceptions. In a few cases secondary growths are found in parts of the bones normally deficient in red marrow. This may be explained by the presence of minute foci of cellular marrow throughout the shafts and epiphyses. It may well be, too, that as a result of the severe constitutional disturbance set up by some forms of malignant disease, an extension of the cellular bone-marrow occurs in order to provide for new blood formation. With this increase in the volume of the red marrow there will be opportunity for secondary deposits to occupy anomalous sites. If we accept Piney's claim that all bone metastases occur in the cellular red marrow, we can then account for the frequency of such deposits in the ribs, vertebræ, skull, sternum, and the upper ends of the humerus and femur. We must explain the exceptional sites of secondary deposits as the result of macroscopic or microscopic irregularities of the distribution of the red marrow. Professor Handley regards the relative freedom of the distal limb-bones from metastases as evidence in favour of the lymphatic permeation theory, contending that the nearer the bone to the primary growth (in this case the breast), the greater the tendency for secondary growths to occur therein. Piney, on the other hand, explains the freedom of the distal limb-bones as the natural outcome of the absence of red marrow from these bones. Systematic examination of the bones in various malignant growths reveals a surprisingly large number of unsuspected metastases, usually very small in size, but all of them situated in the red marrow. Von Recklinghausen[6] believed that when surrounding tissues were also involved, this was due to the central growth having emerged through issuing veins of the bone, and that there was no evidence of the reverse process. Erbslöh and Piney both confirm this opinion.

It appears to me that the evidence is preponderatingly in favour of the claim that bone metastases are blood borne, and that they are due to the lodgement of malignant emboli in the cellular marrow. If we accept the lymphatic permeation theory of the origin of these secondary growths, how can we explain a femoral deposit from carcinoma of the tongue, a metastasis in the humerus from carcinoma of the rectum, a secondary growth in the radius from a primary in the bladder—unless, indeed, we regard these all as exceptional, or, alternatively, consider that the breast metastatizes differently from all other tumours?

PRIMARY TUMOURS WHICH GIVE RISE TO BONE METASTASES.

In order to verify, if possible, the current statements concerning the relative tendencies of neoplasms to produce secondary growths in bones, I have searched the records of the autopsies at the Cancer Hospital from 1888 to October, 1922. It is obvious that the figures which were obtained must suffer from the legitimate criticism that post-mortem examinations, unless specially directed to the examination of the skeleton for metastases, will often fail to reveal anything but the gross and obvious deposits. Only when the bones are systematically removed and sections made with a saw will the smaller secondary growths be discovered, so that the figures in *Table I* can only refer to those more obvious masses easily detected by palpation, by the presence of a spontaneous fracture, or by gross external deformity.

Table I.—Metastases in Bones found in 1144 Autopsies for Malignant Disease, many of them Advanced Cases.

Primary Growth	No. of Cases with Metastases
Carcinoma of breast	34
,, ,, uterus	2
,, ,, œsophagus ..	2
,, ,, thyroid	2
,, ,, gall-bladder ..	1
,, ,, tongue	1
,, ,, prostate	1
,, ,, palate	1
,, ,, adrenal	1
,, ,, kidney	1
Sarcoma of tonsil	1
,, ,, tibia	1
,, ,, testis	1
,, ,, back	1
,, ,, chest wall	1
,, ,, neck	1
Doubtful	1
Total ..	53

This list, while confirming the great frequency of bone metastases in breast carcinoma, does not suggest that the thyroid and prostate are specially prone to such metastases, unless we bear in mind how few of the latter are included among the 1144 cases which came to autopsy. The table does bring out the interesting point that squamous carcinoma (tongue, œsophagus, soft palate) has a tendency to produce secondary deposits in bone, which is not, I believe, generally appreciated.

I have tried to supply the deficiencies in this table of primary tumours by an examination of the bone tumours in the Museum of the Royal College of Surgeons and the museums of the hospitals in London. In addition to those given in the table, I found the following: melanotic sarcoma, sarcoma of the nasopharynx, papilliferous carcinoma of the bladder, hypernephroma,

carcinoma of the renal pelvis, carcinoma of the liver, carcinoma of the rectum, squamous carcinoma of the penis, primary carcinoma of the lung, and carcinoma of the cervix. In confirmation of the statement made above as to the association of squamous carcinoma and bone metastases, there were found preserved in the museums six such specimens, four from the œsophagus and one each from the tongue and penis.

A search of the literature revealed a few other primary tumours not yet mentioned which produce secondary deposits in bone—notably carcinoma of the stomach. The late Sir Norman Moore[14] found one spinal metastasis in 29 autopsies. Colwell,[15] Kurpjuweit,[16] Goetsch,[17] Zade,[18] Perry and Shaw,[19] and Harrington and Kennedy[20] have also recorded such cases, some of them with numerous deposits in bones. Bone metastases have also been noted in sarcoma of the breast by Gross[21] and by Virchow[22], though these statements are open to the objection that malignant tumours of the breast were formerly far more commonly diagnosed as sarcoma than is the case to-day. Glioma of the eye, in addition to the relatively commoner melanotic tumours of that organ, may produce metastases in bone, and Knapp[23] has described one such case in the skull. In deciduoma malignum Roger Williams found 2 examples of bone metastases in 50 cases.

SITES OF ELECTION OF BONE METASTASES.

Table II indicates the extent to which the several bones were affected in the 88 metastases found in 53 cases.

Table II.—Location of Bone Metastases in 53 Cases.

Bone	Number	Per Cent
Vertebræ	19	21·6
Ribs	18	20·4
Sternum	13	14·7
Femur	13	14·7
Skull	9	10·2
Humerus	7	7·9
Pelvis	4	4·5
Tibia	2	2·2
Mandible	1	1·1
Scapula	1	1·1
Clavicle	1	1·1
Total ..	88	

When this list is reviewed in the light of the literature, it appears that there is some difference of opinion as to the bones most commonly involved. Roger Williams,[7] in a collection of reports from several sources, states that in 893 post-mortems in cancer of the breast there were 26·5 per cent with secondary growths in bones, and that the commonest bones involved were the skull (24 per cent) and the vertebræ (19·1 per cent); while Ewing[24] places the sternum, ribs, and femur before the skull and vertebræ in order of

frequency. Kaufmann (quoted by Ewing) found over 50 per cent with metastases in bones in a series of 63 autopsies, 14 per cent of them showing definite osteoplastic changes.

In 238 cases of malignant disease of the thyroid of all types, Ehrhardt[25] found 66 with secondary deposits in bones, a much lower proportion than Kaufmann found for the breast. He places the relative frequency of such deposits in the following order—skull, sternum, spine, ribs, humerus, femur, pelvis. Kaufmann[26] states that in 70 per cent of prostatic carcinomata metastases are found in the bones—a higher percentage than in any other form of malignant disease. Osteoplastic changes he noted in many, and in some it was of a most extensive character, even when the primary growth was so small as to be overlooked until the autopsy.

Adler,[27] in 374 cases of primary malignant disease of the lungs and bronchi, found 57 examples of deposits in the bones (some of them of an extensive nature), with a special tendency to involvement of the ribs, spine, skull, and sternum. The incidence was approximately equal for both sarcoma and carcinoma.

Scudder[28] has collected 17 cases of deposits in bones occurring in hypernephroma. Albrecht[29] mentions 2 of Hochenegg's cases in which the only metastases discoverable at the autopsy were in the bones. Hutchison[30] described a series of suprarenal sarcomata in children in which the initial sign was a tumour of the skull, and found that while the ribs, sternum, and vertebræ were also occasionally involved, the long bones appeared to escape. He admits that some of these might be examples of hypernephromata. Ewing[24] considers that in fully verified adrenal carcinoma, as distinct from hypernephroma, secondary growths in bone are rare; and Hartmann and Lecène[31] found only 1 in 48 cases, though Winkler[32] claims 3 in 10.

There seems to be a tendency for periosteal sarcoma of the femur and tibia to produce deposits in other bones, as Butlin and Colby's[33] figures indicate, and as Greenough's[34] report confirms. That rare tumour of bone, the endosteal endothelioma, has a predilection to metastatize in the flat bones, according to Gideon Wells;[35] though, as Symmers and Vance[35] point out, it may be difficult to distinguish such secondary tumours from multiple primary growths.

The only remaining viscus malignant disease of which has a tendency to produce deposits in the bones is the testis. In view of the obscurity of the classification of such tumours, it is not possible to state the relationship between the exact type of malignant tumour and the tendency to metastases in bones; but of 13 cases of sarcoma of the testis Butlin[37] found 3 with secondary growths in bones.

Nearly all these figures are open to the criticism which has already been mentioned—viz., that in most of these reports a systematic examination of the whole skeleton by sectioning the bones has not been carried out, but only the bones bearing obvious signs of disease have been removed for study. The few observers who have adopted the detailed method of investigation, such as I have outlined, have found that deposits are far more numerous than would be suspected by mere external examination, and that bones generally regarded as immune from metastases may contain minute secondary deposits in the marrow. This is an added reason why the argument for a lymphatic

origin of deposits in bone, based on the alleged immunity of the distal limb-bones, must be rejected.

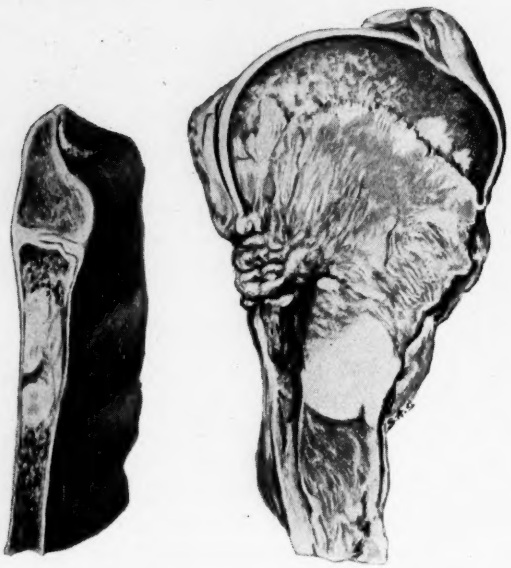


Fig. 20. Fig. 21.

Figs. 20, 21.—Secondary growths in sternum (1), and humerus (2), from case of carcinoma of breast. In (1) the absence of external swelling is noticeable. In (2) there is a spontaneous fracture. *R.C.S. Museum* (*Sir Astley Cooper's Collection*), 2081.1 and 2081.2.

It is difficult to account for the single, slowly growing metastasis which sometimes is found in bones such as the clavicle or scapula, without taking into consideration the traumatic factor. Examples of this kind of deposit are generally associated with obscure primary neoplasms, e.g., of the kidney or thyroid, and a history of trauma is sufficiently common to be noteworthy. Can it be that circulating malignant emboli are able to obtain a footing when the bone is damaged locally by injury? It would help to explain the deposits in the skull in Hutchison's[30] series of adrenal growths in children.

ILLUSTRATIVE CASES.

We will now pass on to a series of cases illustrating the more characteristic features of metastatic tumours of bones, based mainly on specimens from the Museum of the Royal College of Surgeons and the hospital museums in London, and arranged under headings indicating the primary growth.

Breast.—*Figs.* 20 and 21 are from Sir Astley Cooper's Collection, and show metastatic masses in the sternum and humerus. The former exhibits little external deformity, and could easily be overlooked in the usual routine post-mortem examination. The humeral deposit, higher up than is the case with

Fig. 22.—Section from same case as shown in *Figs.* 20 and 21. It has the structure of a rather cellular scirrhous carcinoma of the breast.

many such, is situated where the red marrow of the bone is most abundant. The head is extensively infiltrated, and there is a pathological fracture. The microscopic section (*Fig.* 22), taken from the sternal deposit, reveals the characteristic structure of a scirrhous carcinoma of the breast. The next specimen (*Fig.* 23), from the R.C.S. Museum, illustrates the osteoplastic type of growth, but in spite of this new bone formation there are two fractures,

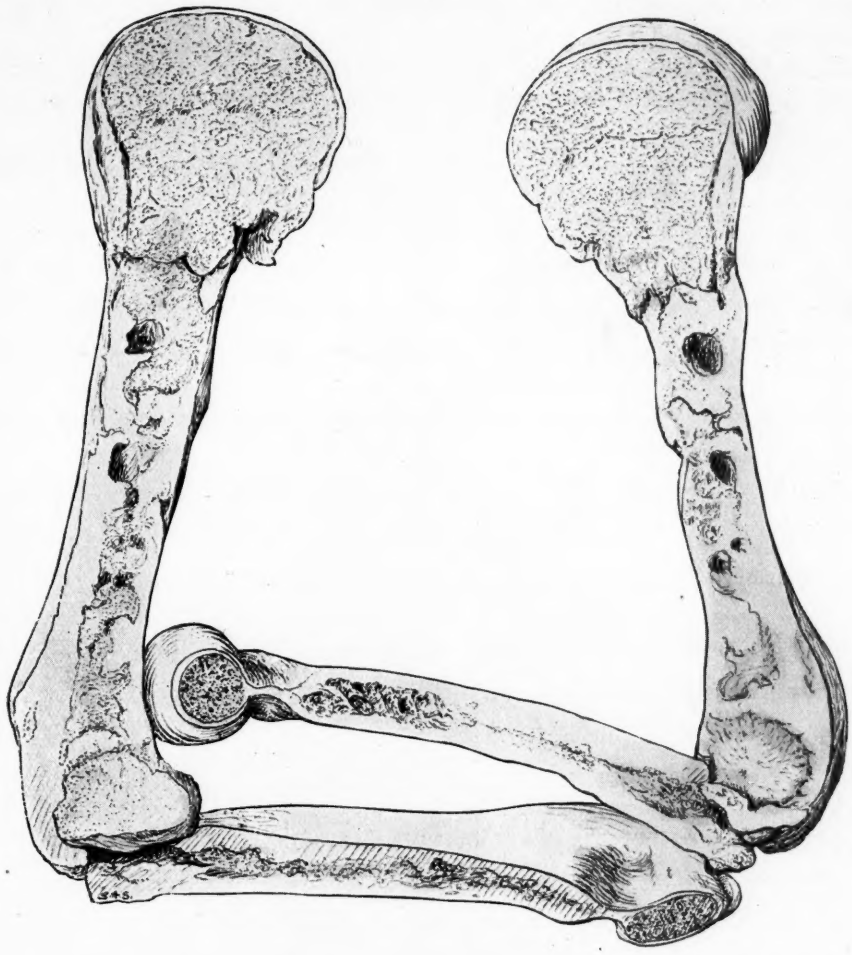


FIG. 23.—Humerus showing extensive infiltration withlsecondary deposit from carcinoma of breast. Much new bone is formed, and there are two spontaneous fractures. *R.C.S. Museum*, 2082.1.

the lower one nearly in the centre of the shaft. There is a specimen in St. Thomas's Hospital Museum (No. 245) of a secondary deposit in the femur, with very little evidence externally of growth, but the whole shaft is infiltrated, and the bone has bent into an **S**-shaped curve. A fracture can be seen in this specimen which has united—not an unusual occurrence in bones with secondary growths of mammary origin.

Thyroid.—The special facility with which malignant disease of the thyroid gland metastatizes in bones has long been recognized. Sir Henry Morris[38] described one of the early cases, associated with a pulsating tumour of the skull. The specimen is preserved in the Museum of the Middlesex Hospital (C. 168). The tumour followed trauma, and was associated with an apparently simple bronchocele. Warrington Haward,[39] soon after, described a case with deposits in the skull, scapula, pelvis, and cervical vertebræ, and of these some pulsated and others did not, indicating that pulsation has no diagnostic significance.

Kanoky[40] states that there is no obvious clinical enlargement of the thyroid in a quarter of the cases of thyroid metastases in bone. In another

Table III.—BONE METASTASES ASSOCIATED

No.	Author	Sex	Age	Site of Metastases	Time Present
1	Cohnheim[41]	F.	35	Right femur, lumbar vertebræ	—
2	Morris, H.[38]	F.	40	Skull, clavicle, both femora ..	2 years ..
3	Haward, W.[39]	F.	59	Skull, pelvis, cervical vertebræ	7 months ..
4	Coats, J.[69]	F.	46	Skull	1½ years ..
5	Litten[71]	—	—	Vertebræ, ribs, pelvis ..	—
6	Feurer[72]	F.	68	Left parietal	9 months ..
7	Gussenbauer[72]	F.	—	10th and 11th dorsal vertebræ	1½ years ..
8	Ewald, C.[73]	F.	45	Scapula	Appeared 1 year after removal of innocent goitre
9	„ „	F.	26	Malar bone	7 years ..
10	Haeckel[70]	F.	48	Lower jaw	3 months ..
11	Kraske[74]	F.	53	Sternum	4–6 weeks ..
12	„	F.	53	Frontal bone	— ..
13	Von Eiselsberg[44]	M.	38	Parietal bone	4 years ..
14	„ „	M.	33	Base of skull	— ..
15	Middeldorpf[75]	F.	56	Skull, vertebræ, sacrum, pelvis, humerus, femur	—
16	Hoffman, K. von[76]	F.	69	Right humerus	—
17	Jaeger, R.[77]	F.	69	Mid-dorsal and lumbar vertebræ	1½ years ..
18	Muzio[78]	F.	43	Pelvis—followed injury ..	—
19	Goebel[47]	F.	54	Right femur	2½ years ..

group the gland, though enlarged, may have undergone no change for thirty years. In yet another group the thyroid may have been operated on for a benign tumour, years before the deposit in the bone appeared. In a majority of cases the metastasis is slow in its growth, and may be the sole metastasis present in the body. This has been confirmed by careful post-mortem examinations. A knowledge of this fact must modify our attitude in treating tumours of bone of thyroid origin.

It was Cohnheim[41] who first claimed that metastasis could occur from a benign enlargement of the thyroid gland, though both von Recklinghausen[42] and Wölfler[43] denied the benign nature of the thyroid in Cohnheim's case. I have arranged in *Table III* the whole series of cases, of which I have been able

WITH A NORMAL THYROID OR BENIGN GOITRE.

Symptoms caused by Metastases	Microscopic Structure of Metastases	Condition of Thyroid Gland	Remarks
—	Innocent goitre ..	General enlargement ..	Autopsy revealed nodule in thyroid penetrating a vein.
Soft pulsating tumour (skull), headache	Normal thyroid gland	General swelling ..	Lived 6 years. Specimen No. C168, the Middlesex Hospital Museum.
Non-pulsating tumour (skull), pulsating (pelvis)	"Ordinary bronchocele"	General swelling present 21 years	Lived 6 *weeks*. Numerous deposits in viscera.
Pulsating, painful ..	Innocent goitre ..	Calcareous change in simple goitre	Goitre present 16 years.
—	—	Adenomatous colloid goitre.	—
Pain and swelling following a blow (3 months)	Typical colloid goitre	Small colloid goitre ..	Recurrence in 10 months.
Pain and paraplegia	Adenoma of thyroid..	Large left-sided goitre..	Goitre not removed.
Swelling	Adenocarcinoma ..	Colloid goitre ..	Microscopically, goitre innocent.
„	Fœtal thyroid—with "slight malignant tendency"	Colloid goitre.. ..	Noticed years *before* goitre.
Pain	Adenoma	Large colloid goitre ..	Goitre not removed, patient alive 3¾ years, no recurrence.
—	Adenoma	Ordinary colloid goitre	Goitre not removed, alive 3 years, no recurrence.
Firm painless swelling	Normal thyroid ..	„ „ „ ..	Still alive after 8 years.
—	Adenoma	Large simple goitre ..	Goitre for 18 years, recurrence 4 years, alive after 8 years.
—	Adenocarcinoma of thyroid	Benign goitre ..	At autopsy, adenomatous nodules in thyroid.
—	Adenoma—typical ..	Small—movable—no sign of malignancy	No change in thyroid throughout.
—	Colloid goitre ..	Simple goitre ..	—
Pain and swelling 6 months after injury..	Normal thyroid—some areas fœtal thyroid	„ „ ..	Goitre present 10 years.
Swelling	Colloid goitre ..	Benign goitre ..	„ „ „
Spontaneous fracture	Benign goitre ..	Moderate-sized goitre—tough	Goitre present 30 years (stationary).

Continued on next page

Table III.—BONE METASTASES ASSOCIATED

No.	Author	Sex	Age	Site of metastases	Time Present
20	Honsell, B.[79]	F.	20	Frontal bone	—
21	Oderfeld, H., and Steinhaus, J.[80]	F.	58	„ „	—
22	Gierke, E.[45]	M.	57	Ribs and dorsal vertebræ ..	4 years
23	Hollis[81]	M.	45	Skull and dorsal vertebræ ..	—
24	Graag, K. S. de[82]	F.	52	Lower dorsal vertebræ ..	—
25	Gierke, E.[45]	M.	46	Dorsal and lumbar spine ..	—
26	Patel[83]	F.	65	Orbit	—
27	Riedel	F.	40	Mandible	7 years
28	Emmerich, E.	M.	63	Sternum, spine, pelvis ..	—
29	Halbron[84]	F.	68	Sternum	—
30	Dercum, F. X.[85]	F.	56	Ribs, cervical vertebræ ..	5 years, 1 year after goitre operation
31	Halpérine[86]	M.	54	Clavicle	20 years
32	Beilby, G. E.[87]	M.	63	Upper jaw	—
33	Kocher, T.[88]	M.	60	Occipital bone	Few months
34	Estor, E., and Massabuaun, G.[68]	F.	40	Left clavicle	5–6 months
35	Guibé and Legueu[90] ..	F.	51	Clavicle	—
36	Jaboulay[89]	M.	77	Humerus	—
37	„	F.	60	Clavicle	—
38	Alamartine, H., and Bonnet, P.[91]	F.	23	Humerus	—
39	Regensburger, F.[46]	F.	55	Humerus	2 years
40	Elmslie, R. C.[63]	F.	61	Radius	3 months
41	Radley and Duggan[48] ..	M.	46	Clavicle	6 months
42	Kanoky, J. P.[40]	F.	40	Skull	6 years before death
43	Knapp, A.[92]	F.	66	Orbit, scapula, ribs	1 week
44	Joll, C. A.	F.	47	Left clavicle	6 months

WITH A NORMAL THYROID OR BENIGN GOITRE—*continued.*

SYMPTOMS CAUSED BY METASTASES	MICROSCOPIC STRUCTURE OF METASTASES	CONDITION OF THYROID GLAND	REMARKS
Non-pulsating tumour	Colloid goitre ..	Simple goitre ..	7 years before, operation cystic tumour (skull); 2½ years before, operation benign goitre.
Swelling	Normal thyroid — as also were all deposits	*No* goitre	Later right lobe enlarged.
Paraplegia ..	Colloid goitre ..	*No* goitre	Small adenoma found at P.M.
„ ..	Thyroid tumour with epithelial proliferation	*No* goitre	—
„ ..	Innocent goitre ..	Colloid goitre ..	—
„ ..	Colloid goitre ..	Small goitre	No clinical evidence of malignancy.
Pulsating tumour ..	Mainly normal thyroid —parts malignant	General enlargement ..	No clinical evidence of malignancy, goitre 30 years, no increase.
—	Normal thyroid ..	No enlargement throughout	Recurrence after 10 years.
—	„ „ ..	—	—
Pulsating tumour ..	Thyroid tissue with malignant epithelial change	Small pulsating goitre ..	Goitre—section—benign colloid type.
Pain and paraplegia ..	—	Simple bilateral goitre	Goitre removed 6 years before.
—	Thyroid tissue ..	General enlargement—no evidence of malignancy	—
—	Thyroid adenoma ..	No trace of goitre ..	—
—	—	Innocent goitre ..	Goitre removed months before.
Pain	Thyroid tissue ..	„ „ ..	No recurrence 1½ years.
Pain and pulsation ..	„ „ ..	Thyroid normal ..	Well 1½ years later.
Pulsating tumour ..	„ „ ..	Small goitre—no symptoms	—
—	„ „ ..	Goitre 7 years—rapid growth 14 months	Sections of goitre—benign.
Pulsating tumour ..	„ „ ..	Benign goitre ..	Death from multiple bone deposits.
Pain	Typical thyroid tissue	Slight general enlargement	Arm disarticulated.
„	Fœtal adenoma ..	Hard fixed swelling right lobe (several years)	X-ray examination, whole skeleton, revealed no other deposits. Well 10 to 12 months later.
Pain and pulsation ..	Secondary carcinoma	Normal in all respects ..	Small thyroid tumour shelled out 2 years before.
Pain and cerebral compression	Normal thyroid tissue	Intrathoracic goitre right lobe	Goitre 20 years before death. Partly removed 9 years after first noticed. No change later in goitre.
Diplopia	Thyroid adenoma ..	No palpable goitre ..	2 years later calcified mass in left lobe of thyroid.
Pain	Normal thyroid tissue	Firm movable tumour right lobe	Well 1 year later.

to obtain records, in which one or more deposits in bone were found associated with what was considered either as a normal thyroid gland or a benign form of tumour or enlargement. That some cases of this character have occurred I think admits of no doubt, since the microscopic structure both of the thyroid gland and of the deposit in bone have been fully investigated, and correlated with the subsequent history of the patient. It is otherwise with the majority of the cases in the list, for either the primary tumour had not been removed or the description of its nature leaves a doubt as to its benignity. In a few of the examples cited in the table the metastatic deposits were clearly malignant in type, even though the primary tumour is described as benign in structure. It seems probable that most of these anomalies can be explained, when the difficulty of drawing the line between innocent and malignant tumours of the thyroid—a difficulty mentioned by nearly all writers on this subject—is appreciated.

FIG. 24.—Vertical section of humerus showing secondary thyroid tumour. *St. Bartholomew's Hospital Museum*, No. 514.

In one of von Eiselsberg's cases,[44] although a secondary deposit in the skull was, after removal, found to have the structure of a benign adenoma, it recurred. He points out, in this connection, that in some cases metastases of bone of thyroid origin have a more innocent microscopical appearance than metastases in such viscera as the lungs. Another of von Eiselsberg's cases illustrated the capacity of a secondary deposit to carry on the function of the thyroid gland, for after removal of such a metastatic mass the patient relapsed into a condition of cachexia strumipriva.

Gierke[45] was able to carry the proof of the thyroid nature of such deposits a step further by the discovery of iodine in them. This author considers such deposits in the bones as essentially malignant, in spite of their apparently benign structure, even when the thyroid is itself normal.

Regensburger[46] describes a case in which the arm was removed for what was thought to be a primary sarcoma, yet on removal this proved to be a thyroid metastasis.

A case somewhat similar, the specimen of which is shown in *Fig.* 24, was presented by the President of the Royal College of Surgeons, Sir Anthony Bowlby, who has kindly provided me with details. This drawing is

taken from the specimen in St. Bartholomew's Hospital Museum (No. 514). The patient, a woman of 53, was said to have had exophthalmos at 28 years of age, but no true evidence that she had Graves' disease is forthcoming. At 38 years the thyroid was examined and found to be enlarged and indurated. It grew no bigger, but became so hard as to appear to be calcified. The thyroid did not change up to the time the arm was removed for a tumour of the humerus associated with pain and disability. The patient lived nearly six years after, but there was evidence in the interval of an intrathoracic extension of the thyroid gland, which was only partly controlled by X-ray treatment. The patient eventually developed a spinal deposit, with spontaneous fracture and paraplegia. The section from the tumour (*Fig.* 25) is clearly of thyroid

FIG. 25.—Section from specimen shown in *Fig.* 24. Well-formed vesicles, some containing colloid, are visible.

origin, with vesicles of irregular size and shape, with a large mass of colloid in one of them, and traces of this material in several others. This case is not included in *Table III* owing to the fact that the subsequent history of the patient suggests a slowly progressive malignant tumour of the thyroid. The great interest lies in the close simulation of a primary tumour of the humerus, associated with an apparently calcified goitre.

Goebel[47] was able up to 1898 to collect 11 cases of metastases of thyroid structure in bones which were treated radically on the assumption that they were primary tumours, and he reported 4 others treated palliatively for various reasons.

In *Table III* it is clear that only a limited number of the cases are reported with that fullness which carries conviction, and in several the thyroid gland

was not operated on at all. Some pathologists maintain that nothing short of a complete survey of the whole thyroid by serial sections could prove the absence of a minute malignant growth, but even this extreme claim cannot prevail against the fact that after the removal of such thyroid metastases the patient has remained well for years and the thyroid has not altered its characters.

My own case is too recent to dogmatize on. The patient, a woman, age 47, was first seen at the Miller General Hospital on Sept. 8, 1921, with a history of pain and weakness in the left arm, and a tumour in the sternal end of the left clavicle. At that time the tumour was firm, but when seen again on Oct. 26 it had increased considerably and there were soft areas in it. A skiagram showed a central tumour with much absorption of bone, and the diagnosis of a myeloma was made.

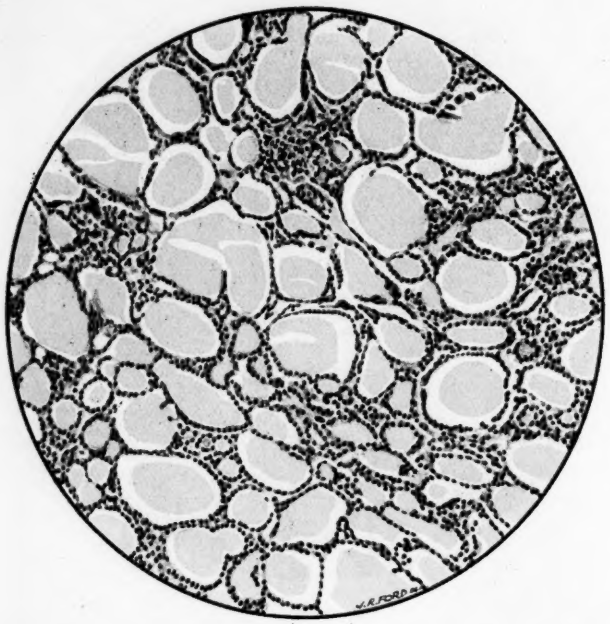


FIG. 26.—Section from tumour of clavicle shown in *Fig.* 27. The regularity of the vesicles, which nearly all contain colloid, clearly indicates an innocent type of tumour.

I operated on Oct. 27, disarticulating the sternal end of the clavicle and freeing it as far out as its middle point, where it was divided and removed with some fibres of the sternomastoid, which it was involving. There was no special difficulty in the operation, and little blood was lost. The wound was drained for a day and healed per primam. The functional result was good, the patient being able to use the arm freely within a month. The sections of the growth were at first thought by Dr. Arthur Davies, Pathologist to the Miller General Hospital, to show a columnar adeno-carcinoma, but on careful re-examination Dr. Davies came to the conclusion that the tumour was of thyroid origin. Sections were cut from five blocks from the whole length of the mass. They all show a similar appearance, which, as may be

seen from the section (*Fig.* 26), is that of an innocent goitre; the vesicles are of regular shape, and most of them contain colloid. Only after the discovery of the thyroid structure of the clavicular tumour was attention paid to the thyroid gland. It was found to contain a small, firm, but quite movable tumour of the right lobe, having all the physical features of an innocent encapsuled tumour. I was not able to persuade the patient to allow me to remove this. A year after the operation there is no change whatever in the size or other character of the thyroid tumour, nor is there evidence of recurrence of the growth. The specimen (*Fig.* 27) has been split longitudinally, and the pale tumour can be seen expanding the bone.

It may be objected in this case that the thyroid tumour may be a malignant adenoma, a growth which resembles closely an innocent thyroid tumour, and is revealed in its true colours only by recurring *in situ* after apparently complete removal. We do not, however, expect to meet with metastases at all in malignant adenoma, but rather do we expect a local recurrence causing death from the involvement of the trachea, etc. It must also be very rare for a malignant adenoma to remain stationary for a year.

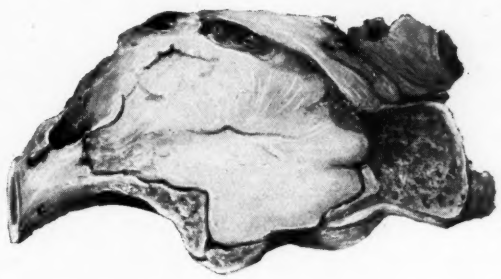


FIG. 27.—Author's case. The sternal end of the clavicle is expanded and absorbed by the pale tumour substance.

Radley and Duggan[48] describe a case of somewhat similar type, operated on by Sir William Thorburn, in which a benign neoplasm had been removed two years before the appearance of the clavicular tumour, and had not recurred at the time of operation on the latter. The structure of the mass was that of a secondary tumour of the thyroid. These authors were so convinced of the integrity of the thyroid itself, that they suggest that the clavicular tumour must be due to a thyroid inclusion in the clavicle of congenital origin. I have submitted this suggestion to Dr. E. Fawcett, Professor of Anatomy at the University of Bristol, who writes: "This inclusion is very unlikely, as the thyroid and the clavicle are developed from different strata, and the thyroid is deep to the depressor muscles of the hyoid."

The relation between the thyroid gland and metastases in bones may therefore be :—

1. The thyroid gland may be quite normal in every way, and the metastasis may have either the structure of normal thyroid tissue, of an innocent thyroid tumour, or of a tumour exhibiting any degree of malignancy.

2. The thyroid gland may be the seat of an innocent diffuse goitre or of an encapsuled innocent tumour, and the bone tumour may have a similar structure. On the other hand, the metastatic tumour may show various grades of malignancy.

3. The tumour of the thyroid may be of any grade of malignancy, yet the metastatic growths in bones may have the structure of an innocent goitre.

Prostate.—These growths have perhaps the greatest tendency of all the primary tumours to produce secondary deposits in bones. In the one example of this in the Cancer Hospital records, the bones affected were the pelvis, ribs, scapula, skull, humerus, and clavicle. A remarkable amount of new bone formation may occur in such deposits, sometimes amounting to a diffuse formation of bone involving the whole shaft, obliterating the marrow cavity, and even projecting from the surface in an irregular series of osteophytes, tubercles, and stalactitiform outgrowths. The new bone may be almost spongy in consistence, or on the other hand extremely dense. Severe secondary anæmia, even simulating pernicious anæmia, may occur if the bone-marrow is extensively replaced by this new bone.

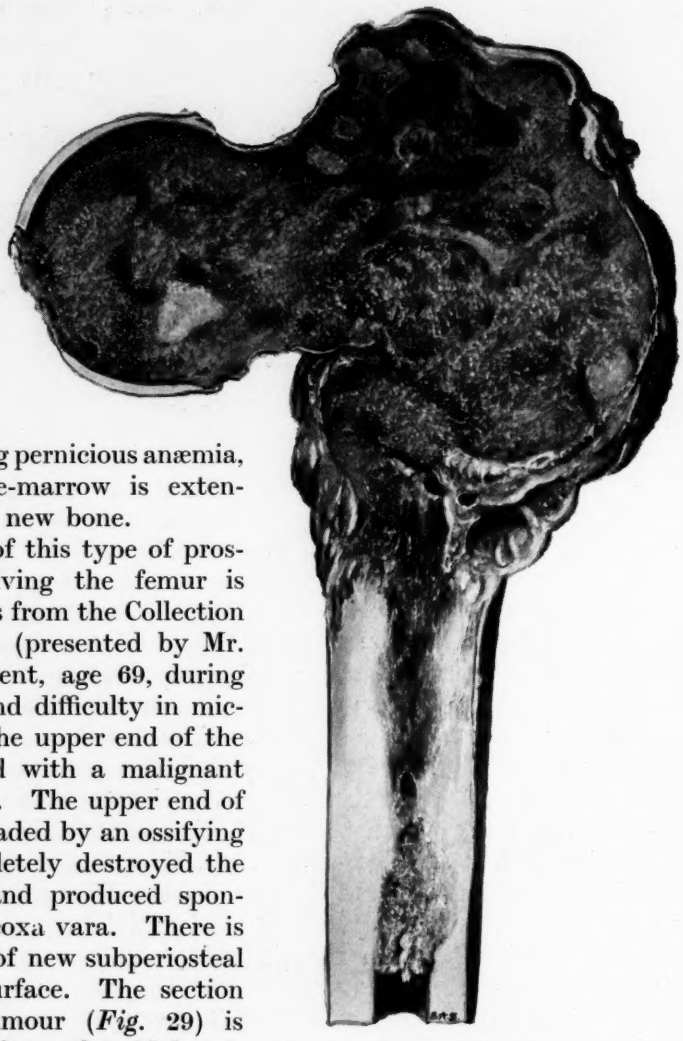


FIG. 28.—Secondary prostatic carcinoma in upper end of femur. Head and neck extensively infiltrated. Spontaneous fracture. Much new bone formation. *R.C.S. Collection* (unmounted).

A good specimen of this type of prostatic metastasis involving the femur is shown in *Fig.* 28. It is from the Collection in the R.C.S. Museum (presented by Mr. Hey Groves), the patient, age 69, during life having had pain and difficulty in micturition, and pain in the upper end of the right femur, associated with a malignant growth of the prostate. The upper end of the femur has been invaded by an ossifying neoplasm, which completely destroyed the normal architecture, and produced spontaneous fracture and coxa vara. There is also an irregular mass of new subperiosteal bone visible on the surface. The section from the primary tumour (*Fig.* 29) is manifestly an infiltrating spheroidal carcinoma, and the secondary deposit (*Fig.* 30) shows a similar structure, with much new bone surrounding the masses of carcinoma cells.

Von Recklinghausen[6] drew attention to the resemblance between this ossifying type of secondary prostatic carcinoma and diffuse chronic inflammatory lesions, and he ascribes to the carcinoma cells themselves the capacity to produce this new bone.

The primary growth in the prostate may occasionally be so small as to be overlooked during life. A specimen in the R.C.S. Museum (No. 1741.1)

FIG. 29.—Section from primary tumour of prostate. The spheroidal carcinoma can be seen invading the bladder muscle fibres. From the case shown in *Fig.* 28.

shows a malignant growth of the prostate which has not caused any enlargement of the gland and gave rise to no symptoms, yet there is a metastatic

FIG. 30.—Section from the secondary deposit shown in *Fig.* 28. Masses of spheroidal cells can be seen in the cavities between the new bone trabeculæ.

deposit in the ribs, the microscopic section of which proves it to be a spheroidal carcinoma similar to that in the prostate.

Another series of specimens in the R.C.S. Museum, from Silcock's case,[49] illustrates that in the same case there may be varying degrees of osteoplastic change, for, while in the deposit in the skull (*Fig.* 31) it is well marked, in the femoral metastasis there is much less new bone, and the ununited pathological fracture shows not a sign of callus. A section clearly exhibits the nature of the primary growth.

Sasse,[50] in discussing the nature of the osteoplastic changes in prostatic metastases, points out that the first deposits are found in the spongy osseous tissue (in other words, where the cellular marrow is found), and adds that this

FIG. 31.—Secondary prostatic carcinoma in skull. There is much new bone formation on both inner and outer surface. The original outline of the skull is clearly preserved. *R.C.S. Museum,* 2091.1.

is only explicable on the theory of conveyance through the blood-stream. Axhausen[51] believes that the osteoplastic property lies in the connective-tissue stroma of the cancerous deposit, but that the stimulus to such ossification proceeds from the carcinoma cell itself. He also emphasizes that osteoclasis goes on side by side with osteoplastic changes; hence spontaneous fractures are quite compatible with a high degree of osteoplastic change in the bone. Erbslöh[13] agrees with von Recklinghausen's opinions on the whole, as to the source of the new bone, but considers that the connective tissue may take a share in the formation of the bony deposits.

Braun,[53] in a series of papers, describes a form of anæmia, closely resembling

pernicious anæmia, associated with the diffuse form of osteoplastic carcinoma of prostatic origin. In the example he cites, the primary growth was too small to be recognized clinically, so that great difficulty arose in distinguishing the tumour of the bone from a primary neoplasm. He adds that the fearful pain in the bones should be helpful in this distinction.

Other Parts of the Genito-urinary System.—The whole of the urinary tract seems to share with the prostate in the tendency to metastases in bones.

The Bladder is here represented by a specimen (*Fig.* 32) from the R.C.S. Museum, presented by John Hilton. An extensive villous papilliferous carcinoma of the bladder can be seen, the infiltrating character of which is displayed in the section (*Fig.* 33). The secondary deposit is in the radius (*Fig* 34), and the microscopic section from this reproduces the transitional papilliferous carcinomatous structure very exactly (*Fig.* 35).

FIG. 32.—Primary papilliferous carcinoma of bladder. The whole of the inner surface is studded with growths. *R.C.S. Museum*, 1780.1 (*presented by J. Hilton*).

FIG. 33.—Section of tumour of bladder shown in *Fig.* 32. A papilliferous infiltrating growth is shown.

FIG. 34.—Deposit in radius secondary to growth of bladder shown in *Fig.* 32. Tumour partly cystic. *R.C.S. Museum*, 2115.1.

FIG. 35.—Section from the deposit in the radius (*Fig.* 34) secondary to tumour of bladder (*Fig.* 32). The papilliferous structure is very strikingly displayed.

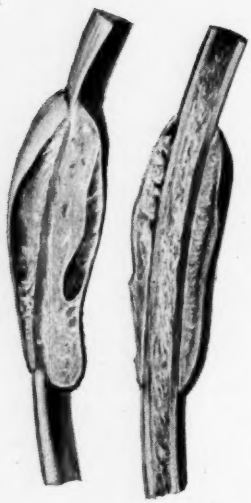


FIG. 36.—Deposit in rib (bisected), showing secondary growth following tumour of renal pelvis shown in *Fig.* 37. *R.C.S. Museum,* 2103.1.

FIG. 37.—A papilliferous carcinoma of the renal pelvis, causing hydronephrosis. *R.C.S. Museum (Hunterian Collection),* 519.

A metastatic deposit in the skull secondary to a bladder growth is preserved in St. Thomas's Hospital Museum (Nos. 2174 and 614, and *see* Clutton[53]), and there is another in the tibia in St. Bartholomew's Hospital Museum (No. 508).

Closely related to this group is the papilliferous transitional-celled carcinoma of the ribs (*Fig.* 36), secondary to a growth in the renal pelvis (*Fig.* 37), both of which specimens are in the Hunterian Collection. The primary growth fills the renal pelvis, and has caused hydronephrosis. The microscopic section (*Fig.* 38) from the metastasis, while confirming the nature of the growth, does not reproduce the papilliferous structure so well as does the metastasis from the vesical growth above.

FIG. 38.—Section of tumour shown in *Fig.* 36. The papilliferous structure is not well marked.

FIG. 39.—Tumour removed from lower end of humerus. The primary growth was a hypernephroma. *R.C.S. Museum*, 2088.1.

The Kidney is well represented among specimens with metastases in bones. One of the most interesting was presented to the R.C.S. Museum by Sir John Bland-Sutton,[54, 55] . . . "a tumour removed by resection from the lower end of the humerus, six years before death" (*Fig.* 39). It was originally thought to be a primary neoplasm. *Fig.* 40 shows the right kidney and adrenal from the same patient, removed after death. The primary growth appears from Dr. Shaw Dunn's examination to be a hypernephroma, and the adrenal growth is apparently, like the humeral deposit, a metastasis. There are several such cases on record. In St. Mary's Hospital is a clavicle containing a deposit from a hypernephroma removed by Mr. V. W. Low[56] (and referred to by him in the discussion on "Secondary Growths in Bone" at the Royal Society of Medicine

in 1920). Mr. Cope has kindly informed me of a similar case of his own, involving the humerus. Dr. Nicholson[56] has described a deposit in the tibia secondary to an embryonal tumour of the kidney. Mr. Nitch[56] described two cases of adrenal growths with secondary deposits in the humerus and forearm

FIG. 40.—Right adrenal and kidney from same case as *Fig.* 39. The adrenal mass is, like the humeral one, secondary to a hypernephroma. *R.C.S. Museum*, 1736.1.

bones respectively, in both of which the secondary tumour was detected before the primary lesion. Winkler[32] refers specially to the tendency of adrenal neoplasms to remain for long periods symptomless, so that the metastases they so often produce in the bones are treated as primary tumours. He also calls attention to the way in which these tumours invade the renal

vein, and thus eventually the inferior vena cava, whence emboli readily reach the right side of the heart. There is a good specimen in the London Hospital Museum (No. 684A) of that variety of adrenal tumour which occurs in children and (as Hutchison[30] describes) gives rise to metastases, especially in the skull.

Testicular Tumours also may have secondary deposits in the bones. There is a preparation in University College Hospital Museum, from a case under the care of Mr. Barrington, in which the primary tumour is soft and yellow from necrosis, with some fibrous trabeculæ dividing it into lobes. It does not invade the spermatic cord. Microscopically it is composed of cells, arranged in alveoli and containing lumina. The cells stand in many places directly on capillary walls. The secondary growths in the lungs resembled large round-celled sarcoma, but the growth is described as probably an endothelioma. The deposit in the spine involves the eleventh dorsal to the fourth lumbar vertebræ. The brownish hæmorrhagic growth has invaded the spinal canal and destroyed the body of the first lumbar and a good deal of the second lumbar and twelfth dorsal vertebræ. The patient, age 40, had had for nine years before death an enlarging left testicle, following a kick from a horse. Paraplegia and other evidences of the spinal deposit appeared three and a half years before death, and two years before that event the spine fractured spontaneously. There is also a specimen in the same museum (No. 78R) of a secondary deposit in a rib, from a primary epitheliomatous growth of the penis.

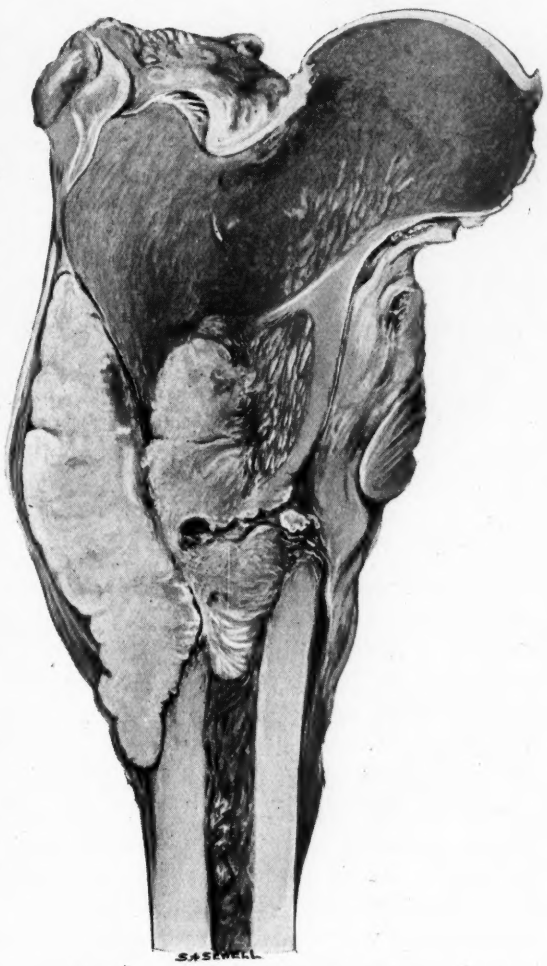


FIG. 41.—Deposit in upper end of femur secondary to carcinoma of tongue. There is a spontaneous fracture. *R.C.S. Museum*, 2108.1.

The Female Generative Organs provide a number of examples of tumours which metastatize in the bones, although I have been unable to find a single specimen of a primary growth in the ovary in this category. In the Museum of the R.C.S. is a preparation which shows a mass of growth in the parietal bone, projecting considerably from its outer surface, and, to a slight

extent, from the inner. Microscopically this is a spindle-celled sarcoma, which appeared twelve months before death in a patient, age 54, who had had hysterectomy done six months before the cranial tumour was noticed. In University College Hospital there is a specimen (No. 78K) of a deposit in the right humerus, with a spontaneous fracture, secondary to a carcinoma of the cervix. Like many of the specimens referred to in this paper, it was removed under the impression that it was a primary growth, the swelling having appeared while the uterine symptoms were still trivial.

FIG. 42.—From the deposit shown in *Fig.* 41. The structure is that of a rapidly growing squamous carcinoma.

Tongue.—The tongue is not generally believed to give rise to distant metastases. There is such a case on record among those reviewed from the Cancer Hospital post-mortem series, but I can find no details of the case, and the specimen has been lost. *Fig.* 41, from a preparation in the Museum of the R.C.S., shows a right femur with a spontaneous fracture due to a secondary squamous carcinoma, the primary mass being in the tongue. The microscopic section of the secondary growth (*Fig.* 42) reveals a rapidly-growing epithelioma with cell-nests and obvious prickle cells. Dr. Shaw, Pathologist to the Royal Northern Hospital, has kindly supplied me with details of the post-mortem findings. In addition to a large mass in the tongue and floor of the mouth, the glands from the jaw to the bronchi were involved, as well as the pleura, lungs, abdominal wall, small intestine, liver, and femur.

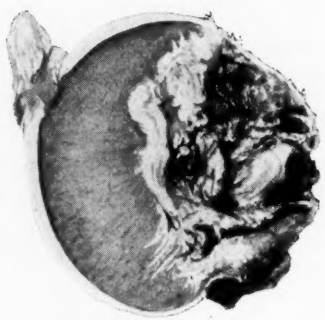


FIG. 43.—Metastatic deposit in head of femur secondary to carcinoma of œsophagus. *R.C.S. Museum,* 2110.1.

Œsophagus.—There are several examples in the London museums of bone deposits secondary to carcinoma of the œsophagus. The one illustrated (*Fig.* 43) is from the R.C.S. Museum, and involves the femur. The microscopic section of this metastasis (*Fig.* 44) fully confirms the nature of the primary lesion. There are two preparations in St. George's Hospital Museum (Nos. 73D and E) of metastases in the femur, and in the

pelvis and ribs (Nos. 73K and L), also secondary to carcinoma of the œsophagus, and a third in the Westminster Hospital Museum of the same sort in the ribs.

Alimentary Canal.—I have been unable to find a specimen in the museums in London of metastases in bones secondary to carcinoma of the *stomach*, though Colwell[15] found 5 examples in 227 cases—the bones affected being ribs, vertebræ, humerus, and sacrum. In other recorded cases osteoplastic changes seem to be not uncommon, and perhaps as a consequence profound anæmia may be a prominent feature. I have also failed to find a tumour of the small bowel with metastases in the bones, but the *large gut* certainly has this tendency. Rowntree has described a deposit in the skull from a carcinoma of the sigmoid colon,[56] and there are several specimens associated with carcinoma of the rectum. One such preparation is in St. Thomas's Hospital Museum (No. 677A), a humeral growth in a patient, age 32. Two fractures are present, and the growth coincides very closely in position with the normal distribution of the red marrow in the humerus. The case has been described by Pitts.[57] The microscopic section (*Fig.* 45) is from

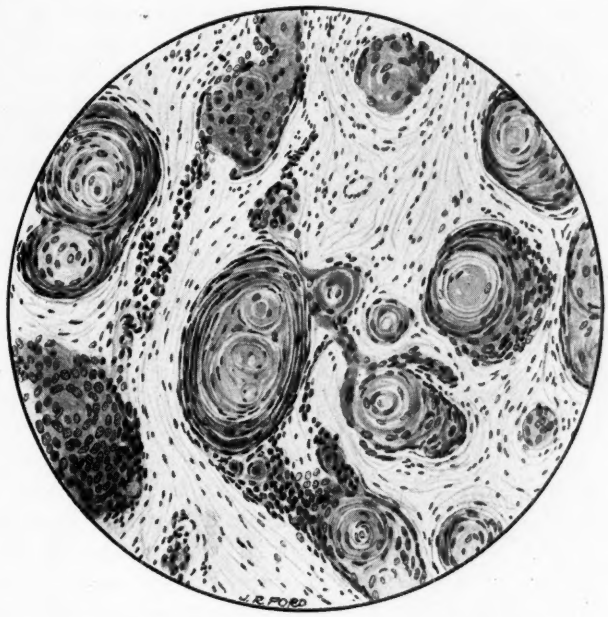


FIG. 44.—From specimen shown in *Fig.* 43. The 'cell-nests' are well shown.

FIG. 45.—Section from deposit in humerus secondary to carcinoma of rectum. The adeno-carcinomatous structure is unmistakable. *St. Thomas's Hospital Museum*, No. 677A.

a preparation made by Professor Shattock, and reveals the structure of a typical adeno-carcinoma such as commonly occurs in the rectum. *Fig.* 46 shows a deposit in the upper end of the sternum secondary to a similar rectal growth, from a patient, age 35, who had also a deposit in the spine. The specimen is in the University College Hospital Museum (*No.* 78J). In the *Stethoscope*[58] for December, 1922, a metastasis in the ulna is described, secondary to carcinoma of the rectum. Amputation was performed for the pain and disability, with at least temporary benefit.

FIG. 46.—Metastasis in upper end of sternum secondary to carcinoma of rectum. *University College Hospital Museum*, 78.1.

Liver.—Primary growths of the liver occasionally metastatize in bones. The specimen from the Museum of the R.C.S. shown in *Fig.* 47 is from a case reported by Dr. Newton Pitt.[59] The secondary mass in the spine is interesting because although there is a striking degree of angular deformity, due largely to the destruction of one vertebral body, there was no sign of involvement of the cord. The microscopic section (*Fig.* 48) is from the spinal deposit, and has an irregularly formed glandular structure, with indications of a columnar type of cell. The primary mass was in the liver.

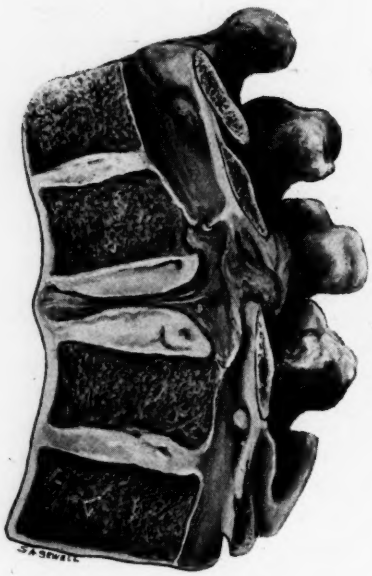


FIG. 47.—Deposit in spine secondary to primary growth in liver. Angulation due to destruction of body of one vertebra is well seen.
R.C.S. Museum, 2100.1.

FIG. 48.—From specimen shown in *Fig.* 47. The adeno-carcinomatous structure is similar to that of the primary growth in the liver.

Respiratory Tract.—There are one or two metastases from the respiratory tract worthy of note. *Fig.* 49, from the Museum of the R.C.S., shows many deposits of varying size over the whole skull. It is clear that these, when small, started in the diploë, for only the larger ones have perforated both tables. A section taken from one of these deposits has the structure of a small round-celled sarcoma. The patient, a man of 45, had had a sarcomatous polypoid mass in the nasopharynx for a year before the skull tumours appeared, and, later, many other bones, including even the metacarpals and phalanges, became the sites of metastases. In the Museum of Westminster Hospital

FIG. 49.—Numerous secondary deposits in skull. The primary growth was in the naso-pharynx.
R.C.S. Museum, 2052.1.

(No. 265A) there are specimens showing deposits in the femur, ribs, and vertebræ from a primary growth of the right lung and pleura.

Bones.—It is natural to assume that primary growths of the bones would produce deposits in other bones, and, in actual fact, this is sometimes the case. A preparation in the Museum of the R.C.S. shows a growth secondary to an osteoid sarcoma of the femur in a boy, age 9. The temporal metastasis also exhibits osteoid changes, and a microscopic section taken from the metastasis illustrates the sarcomatous structure; the osteoid changes are pronounced. This case was described by Durham.[60] In the Westminster Hospital Museum are similar ossifying growths in the skull, pelvis, and rib secondary to an ossifying sarcoma of the femur (Nos. 263A, B, C, D, and E).

Melanotic Sarcoma.—Melanotic sarcoma, with its remarkable power of dissemination, does not spare the bones. The specimens illustrated in *Figs.* 50, 51, and 52 are from the Museum of the R.C.S., and the growths are in the

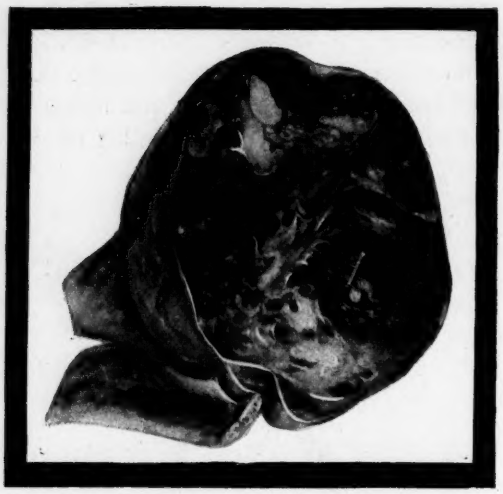


FIG. 50.—Melanotic growth in ribs secondary to primary in thumb. *R.C.S. Museum*, 2066.1.

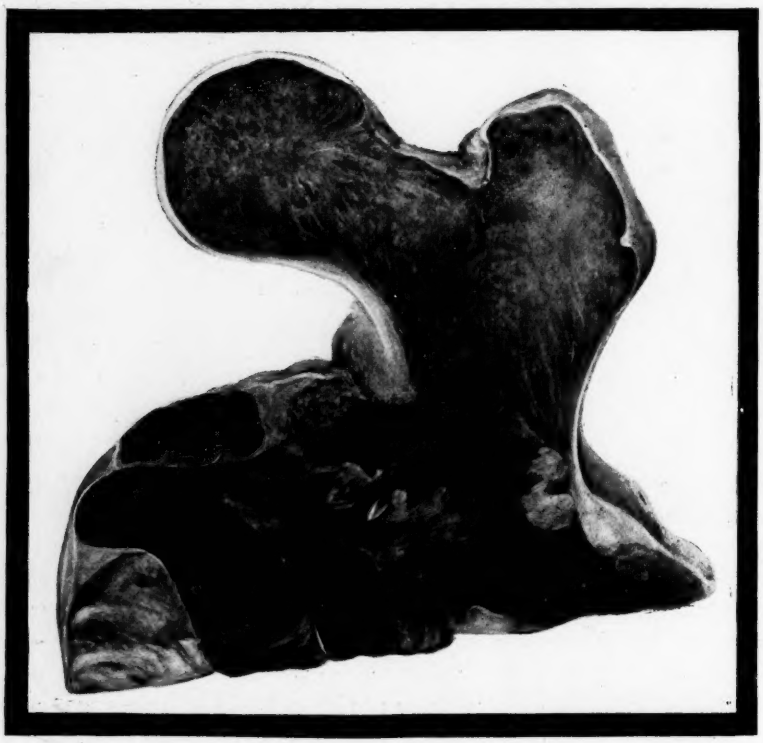


FIG. 51.—From same case as *Fig.* 50. Deposit in upper end of femur. Primary growth was in thumb. *R.C.S. Museum*, 2066.2.

ribs and the upper end of the femur. The primary growth was in the thumb, and there were other secondaries in the glands, liver, and humerus. All the secondary growths are well pigmented, though this is by no means always so. Another series of preparations in the Museum of George Longstaff, Esq., includes deposits in the ribs, sternum, and dorsal vertebræ. The primary lesion was a pigmented mole on the shoulder, which took on active growth five months before death. In contrast to this rapid dissemination, Cairns[61] reports a recurrence in the scapula 18 years after the removal of the left eye for a melanotic tumour.

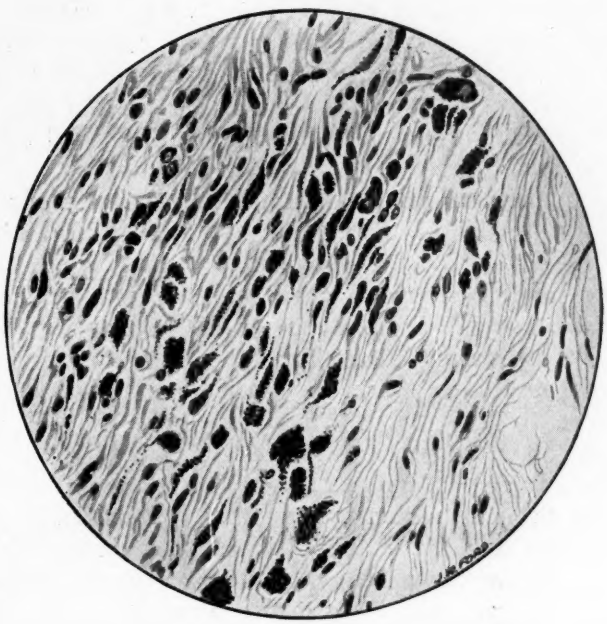


FIG. 52.—From same case as *Figs.* 50 and 51. The pigmented cells are somewhat spindle-shaped.

DIAGNOSIS.

As a rule diagnosis should be a simple matter, because evidence of the primary tumour will generally be available. On the other hand, deeply placed primary neoplasms in stout patients may be missed even when special attention is directed to this possibility. The kidney and adrenal provide good examples of this difficulty. Of the five cases of renal and adrenal neoplasm with metastases in bones mentioned at the discussion at the Royal Society of Medicine in 1920, the latter were in every case treated as primary lesions because of the obscurity of the primary growths. In Mr. Cope's case, which he has kindly allowed me to mention, a growth of the humerus was treated by amputation in a man of 72, and sections proved it to be a hypernephromatous tumour. No evidence of the primary growth could be obtained by palpation or by skiagraphy.

It is proverbially easy to be wise after the event, yet in most cases such as those just mentioned it seems unlikely that any method of investigation could have revealed the primary growth. A hypernephroma may grow very slowly in some cases, yet it may give rise to a secondary deposit, perhaps single, in some remote part, while the primary tumour is still too small to be felt, or detected by radiography. Delbet[62] asserts that, even after removal, secondary carcinomata of bones may pass as sarcomata because the epithelial elements may be few in number. He reports a tumour of the olecranon which he removed as a primary growth; this opinion, while at first confirmed, was subsequently upset by the discovery of a small mass of epithelial cells in the section, and eventually the primary mass was traced to the kidney. Delbet

claims that clinically the distinction between a primary and a secondary tumour of a bone may be almost impossible, especially if the epiphyseal region be involved. Secondary tumours, he says, affect the shaft so much more often than primary tumours that this point has some diagnostic value.

Pain is stated by Delbet to be an unusual symptom in secondary tumours of bone, though it is often early and prominent in primary neoplasms. Elmslie[63] maintains on the contrary that early occurrence of pain is in favour of the tumour being metastatic. Harrington and Kennedy[20] regard pain and tenderness in the bones as highly suggestive of deposits in the bone-marrow.

When the secondary deposit is in the spine, Oppenheimer[64] states that *deformity* may be the first thing noticed, and this is borne out by Pitt's case, to which reference has already been made.

The *osteoplastic tendency* shown particularly by prostatic metastases, and to a less extent by mammary, gastric, thyroid, and gall-bladder secondary deposits, at once distinguishes them from most primary neoplasms, except the ossifying type of periosteal sarcoma, but, as von Recklinghausen[6] says, they may still be mistaken for diffuse osteoperiostitic lesions. A Wassermann test may help to eliminate the latter, though as there are other causes of chronic diffuse inflammation of bone besides syphilis, the differentiation may be extremely difficult.

It is almost a platitude to say that the prostate and thyroid should be examined with minute care. A tumour in either, which by itself would give rise to little anxiety, may have a special significance when associated with a bone tumour of doubtful nature. There still remain a few cases in which the prostate on the one hand or the thyroid on the other is apparently normal to all tests, yet deeply placed within there is a small malignant nodule, which can only be discovered at autopsy.

Spontaneous fractures occur in both primary and secondary tumours of bone, but in the former the tumour has generally been noticed before the fracture, while with the latter fracture may be the first thing and the tumour is then detected ; but in a few there is little evidence externally of a neoplasm at any stage. Union of such fractures is much more likely to follow when the growth is a secondary deposit than when the fracture is associated with a primary sarcoma, this difference being possibly related to the osteoplastic properties of some secondary growths.

I have already mentioned that *anæmia* of a severe degree may be a feature of metastatic tumours of bones, and it has been observed before the deposits in the osseous system became evident—even before the primary tumour. In these rare cases the differential diagnosis depends on such fine distinctions in the blood examination as Piney has referred to in his paper, which must be consulted for details.

Radiography.—X-ray investigation should help materially in the diagnosis of these growths. Bloodgood[65] holds that the distinction from a primary tumour can be made as a rule, because in the radiogram there is evidence both of bone destruction and of bone formation in the central shadow, while, except in a healing bone cyst, this is never found in primary growths. He, however, admits the difficulty with secondary tumours of bone of prostatic origin, which so closely resemble—even radiographically—ossifying primary sarcomata.

Elmslie says that where the long bones are involved, a slight expansion is seen in the radiogram, the clear area which lies within giving the appearance of rarefied bone which shades off gradually up and down the shaft into the normal bone. It is this lack of definition of the tumour that he regards as characteristic. Baetjer and Waters[66] take up very much the same position, stating that it is impossible in the radiogram to determine the point where the normal bone ends and the growth begins. These authors state that in the osteoplastic type of metastasis new bone is laid down only at the periphery of the growth; but that this is not so can be seen in the radiogram of the prostatic deposit in the femur from the case I have already mentioned. Nichols[67] goes still further, saying that in bone carcinomata evidence of new bone formation is entirely lacking. Greenough, Simmons, and Harmer[34] state that any metastatic tumour may simulate very closely the radiographic picture of a primary sarcoma, since perpendicular spicules are not always present in the latter and not invariably absent from the former.

TREATMENT.

Treatment will but seldom be undertaken if the true secondary nature of the tumour be recognized, though severe pain, or the imminence of fungation, may occasionally justify amputation. It is perhaps a counsel of perfection to suggest that all bone tumours should be explored and examined microscopically before any radical operation is undertaken, but it would prevent the more heroic measures when local operations would be alone justifiable. The beneficial results of such local resections is very well shown by Sir John Bland-Sutton's case. The patient was able to follow his practice as a medical man for nearly six years after the operation, which he could hardly have done had amputation been chosen.

PROGNOSIS.

As a rule—and this is well brought out in *Table III*—the benefit to be expected from operations on secondary growths of the bones is but transitory; recurrence either locally or in other bones, etc., must soon end life. There are a few striking exceptions. Sir Anthony Bowlby's patient lived nearly six years after the removal of the arm for a secondary thyroid tumour of the humerus. Kraske's patient (*Table III*) was alive eight years later and free from recurrence. Riedel's patient (*Table III*) was alive ten years after operation, but recurrence was then obvious. Estor and Massabuaun's[68] patient was alive without recurrence fifteen months after the removal of the thyroid tumour of the clavicle; my own patient is well without recurrence a year after the operation. Elmslie's thyroid tumour of the radius was well for ten months and then was lost sight of. It would seem, therefore, that when the exploration of the growth reveals a secondary tumour of thyroid or renal origin, it is a reasonable course to carry out a limited resection of the bone involved where this is feasible, and that in a few instances amputation is justifiable.

I have great pleasure in acknowledging my indebtedness to Sir Anthony Bowlby, President of the R.C.S., who has kindly given details of his case shown

in *Fig.* 24; to Sir John Bland-Sutton for his help in the case of secondary hypernephroma of the humerus; to Professor Shattock for the section of the rectal carcinomatous growth in the humerus, and for help in other ways; to Mr. C. E. Shattock for assistance with the selection of specimens; to Dr. Davies, Pathologist of the Miller Hospital; to Dr. Shaw, Pathologist to the Royal Northern Hospital; to Dr. Shaw Dunn; to Dr. Piney; to the Curators of the Museums of the Hospitals in London for giving me access to specimens and for permission to have preparations drawn; to Dr. Leitch for permission to use the Cancer Hospital records; to Dr. Knox for help with the radiographic diagnostic features; and to Mr. Sewell and Mr. Ford for their care with the drawing of the macroscopic and microscopic preparations respectively.

BIBLIOGRAPHY.

[1] HANDLEY, W. S., *Cancer of the Breast and its Treatment*, 2nd ed., 1922.
[2] PINEY, A., "Carcinoma of the Bone-Marrow", *Brit. Jour. Surg.*, 1922, x, 235.
[3] PINEY, A., "The Anatomy of the Bone-Marrow", *Brit. Med. Jour.*, 1922, ii, 792.
[4] ROGER, H., and JOSUÉ, O., *La Moelle osseuse*, Paris, 1899.
[5] DEWEY and NOYES, "Lymphatics of the Teeth", *Dental Cosmos*, xlix.
[6] VON RECKLINGHAUSEN, F., "Die fibröse oder deformierende Osteitis, die Osteomalacia, und die osteoplastische Carcinome in ihren gegenseitigen Beziehungen", *Festschrift Rudolf Virchow* 71 *Geburstage*, Berlin, 1891.
[7] WILLIAMS, R., *Natural History of Cancer*, London, 1908.
[8] SCHMORL, *Verschleppung der Chorionzotten*, Leipzig, 1893.
[9] VEIT, J., "Ueber Deportation von Chorionzotten", *Zeits. f. Geburtsh. u. Gynäkol.*, 1901, xliv., 466.
[10] GOLDMANN, E., "Allgemeinen Gefassaufbau malignen Tumoren", *Deut. med. Woch.*, 1906, xxxii, 1683; "Growth of Malignant Disease in Man and the Lower Animals", *Lancet*, 1907, ii, 1236.
[11] LEAF, C. H., "On the Relation of Blood to the Lymphatic Vessels", *Lancet*, 1900, i, 606.
[12] SCHMIDT, M. B., *Die Verbreitungswege der Karzinome*, Jena, 1903.
[13] ERBSLÖH, W., "Fünf Fälle von osteoplastischen Carcinome", *Arch. f. pathol. Anat.*, 1901, clxiii, 20.
[14] MOORE, N., "On the Distribution and Duration of Visceral New Growths", *Lancet*, 1889, ii, 415.
[15] COLWELL, H., "Primary Malignant Disease of the Stomach: a Statistical Study, based upon the Records of the Middlesex Hospital", *Arch. Middlesex Hosp.*, 1906, vii, 151.
[16] KURPJUWEIT, O., "Zur Diagnose von Knochenmarkmetastasen bei malignen Tumoren aus dem Blutbefunde", *Deut. Arch. f. klin. Med.*, 1903, lxxvii, 552.
[17] GOETSCH, W., "Ueber den Einfluss von Knochenmetastasen auf das Knochengewebe", *Beitr. z. pathol. Anat.*, 1906, xxxix, 218.
[18] ZADE, M., "Ein Fall von primärem Magenkarcinom mit nahlreichen Skeletmetastasen und Stauungspapille", *Beitr. z. pathol. Anat.*, 1904–5, xxxvii, 568.
[19] PERRY, E. C., and SHAW, L. E., "An Examination of 50 Cases of Malignant Disease of the Stomach", *Guy's Hosp. Rep.*, 1891, xlviii, 155. (*Case* 21.)
[20] HARRINGTON, A. W., and KENNEDY, A. M., "Bone-Marrow Metastases and Anæmia in Gastric Cancer", *Lancet*, 1913, i, 378.
[21] GROSS, S. W., "Sarcoma of the Female Breast", *Amer. Jour. Med. Sci.*, 1887, xciv, 17.
[22] VIRCHOW, R., *Pathologie des Tumeurs*, Vol. II, 360, Paris, 1869.
[23] KNAPP, *Die intraocularen Geschwülste*, 1868.
[24] EWING, J., *Neoplastic Diseases*, 2nd ed., 1922.
[25] EHRHARDT, O., "Zur Anatomie und Klinik der Struma maligna", *Beitr. z. klin. Chir.*, 1902, xxxv, 343.
[26] KAUFMANN, "Die Verletzungen und Krankheiten der Prostata", *Deut. Chirurgie*, 1902, liii, 450.
[27] ADLER, I., *Primary Malignant Growths of the Lung and Bronchi*, New York, 1912, 62.

[28] SCUDDER, C. L., "The Bone Metastases of Hypernephroma", *Pubs. Massachusetts Gen. Hosp.*, 1907, i, No. 3, 82.
[29] ALBRECHT, P., "Beiträge zur Klinik und pathologischen Anatomie der malignen Hypernephrome", *Arch. f. klin. Chir.*, 1905, lxxvii, 1073.
[30] HUTCHISON, R., "On Suprarenal Sarcoma in Children, with Metastases in the Skull", *Quart. Jour. Med.*, 1907–8, i, 33.
[31] HARTMANN, H., and LECÈNE, *Travaux de Chirurgie*, 1904.
[32] WINKLER, *Die Gewächse der Nebennieren*, Jena, 1909.
[33] BUTLIN, H. T., and COLBY, F. E. A., "On Sarcoma of the Bones of the Leg and Thigh", *St. Bart.'s Hosp. Rep.*, 1895, xxxi, 30.
[34] GREENOUGH, R. B., SIMMONS, C. C., and HARMER, T. W., "Bone Sarcoma", *Jour. of Orthop. Surg.*, 1921, xix, 602.
[35] WELLS, H. G., "The Relation of Multiple Vascular Tumours of Bone to Myeloma", *Arch. of Surg.*, 1921, ii, 435.
[36] SYMMERS, D., "The Multiple Myelomata and their Ability to Metastasize", *Ann. of Surg.*, 1918, lxvii, 687.
[37] BUTLIN, H. T., *Sarcoma and Carcinoma*, 1882.
[38] MORRIS, Sir H., "Pulsating Tumour of the Left Parietal Bone, Right Clavicle, and both Femora", *Trans. Pathol. Soc. Lond.*, 1880, xxxi.
[39] HAWARD, W., "Case of Bronchocele, with Secondary Growths in Bones and Viscera", *Trans. Pathol. Soc. Lond.*, 1882, xxxiii, 291.
[40] KANOKY, J. P., "Thyroid Tumours of Bones", *Surg. Gynecol. and Obst.*, 1916, xxii, 679.
[41] COHNHEIM, J., "Einfacher Gallertkropf mit Metastasen", *Arch. f. pathol. Anat.*, 1876, lxviii, 547.
[42] VON RECKLINGHAUSEN, F., "Bemerkungen zu dem Aufsatz des Herrn. Cohnheim ('Einfacher Gallertkropf mit Metastasen')", *Arch. f. pathol. Anat.*, 1877, lxx, 153.
[43] WÖLFLER, A., "Zur chirurgischen Behandlung des Kropfes", *Arch. f. klin. Chir.*, 1883, xxiv, 157.
[44] VON EISELSBERG, A. F., "Ueber Knochen-metastases des Schilddrüsenkrebses", *Verhandl. Deut. Gesell. f. Chir.*, 1893, xxii, 255.
[45] GIERKE, E., "Ueber Knochentumoren mit Schilddrüsenbau", *Arch. f. pathol. Anat.*, 1902, clxx, 464.
[46] REGENSBURGER, F., "Schilddrüsenmetastasen im Knochen", *Berl. klin. Woch.*, 1912, xlix, 1560.
[47] GOEBEL, C., "Ueber eine Geschwulst von schilddrüsenartigem Bau im Femur", *Deut. Zeits. f. Chir.*, 1898, xlvii, 348.
[48] RADLEY, S. B., and DUGGAN, W., "A Case of Thyroid Tumour of the Clavicle treated by Operation", *Brit. Jour. Surg.*, 1913–14, i, 401.
[49] SILCOCK, A. Q., "Cancer of the Prostate with Secondary Ossific Deposits in the Cranium and Femur", *Trans. Pathol. Soc. Lond.*, 1884, xxxv, 244.
[50] SASSE, F., "Ostitis carcinomatosa bei Carcinom der Prostata", *Arch. f. klin. Chir.*, 1894, xlviii, 593.
[51] AXHAUSEN, G., "Histologische Studien über die Ursachen und den Ablauf des Knochenumbaus im osteoplastischen Karzinom", *Arch. f. pathol. Anat.*, 1909, clxxxv, 358, 459.
[52] BRAUN, L., "Ueber osteoplastisches Carcinom der Prostata zugleich ein Beitrag zur Genese der perniciosen Anämie", *Wien. med. Woch.*, 1896, xlvi, 480, 527, 583.
[53] CLUTTON, H. H., "Alveolar Sarcoma of Skull, Secondary Growth of Bladder", *Trans. Pathol. Soc. Lond.*, 1883, xxxiv, 212.
[54] BLAND-SUTTON, Sir J., "Spolia Opima", *Brit. Med. Jour.*, 1918, ii, 593.
[55] BLAND-SUTTON, Sir J., *Tumours*, London, 1922.
[56] BLAND-SUTTON, Sir J., LOW, V. W., NITCH, C. A. R., NICHOLSON, G. W., ROWNTREE. C., "Discussion on Secondary Deposits in Bone Mistaken for Primary Tumours", *Proc. Roy. Soc. Med.* (Surg. Sect.), 1919–20, xiii, 1.
[57] PITTS, B., "Two Cases of Hernia Complicated by Tubercular Implication of Sac and Contents", *Trans. Pathol. Soc. Lond.*, 1892, xliii, 267.
[58] "Columnar Carcinoma of the Humerus Secondary to Tumour of Upper Part of Rectum", *Stethoscope*, 1922, xx, 66.
[59] PITT, G. N., "Primary Scirrhus of Liver of Tubular Type, with Secondary Growths in Liver, Lung, Vertebra, etc.", *Trans. Pathol. Soc. Lond.*, 1887, xxxviii, 151.
[60] DURHAM, A. E., "Enormous Osteo-sarcoma of Left Femur in a Boy", *Trans. Pathol. Soc. Lond.*, 1883, xxxiv, 263.
[61] CAIRNS, F. D., "Metastatic Melanoma of Scapula", *Brit. Jour. Surg.*, 1922–23, x, 290.

[62] DELBET, P., "Sur les Tumeurs secondaires des Os", *Bull. de l'Acad. de Méd.*, 1920, 3rd s., lxxxiv, 231.
[63] ELMSLIE, R. C., "The Diagnosis of Endosteal Tumours", *St. Bart.'s Hosp. Rep.*, 1912, xlviii, 73.
[64] OPPENHEIMER, E. D., "Early Symptoms of Spinal Cancer", *Jour. Bone and Joint Surg.*, 1922, iv, 342.
[65] BLOODGOOD, J. C., "Bone Tumours, Benign and Malignant", *Amer. Jour. Surg.*, 1920, xxxiv, 229.
[66] BAETJER, F. H., and WATERS, C. A., *Injuries and Diseases of the Bones and Joints*, London, 1921, 243.
[67] NICHOLS, B. H., "Radiographic Diagnosis of Bone Tumours", *Surg. Gynecol. and Obst.*, 1922, xxxv, 301.
[68] ESTOR, E., and MASSABUAUN, G., "Les prétendus Kystes branchiogènes multiloculaires du Cou", *Rev. de Chir.*, 1908, xxxviii, 341.
[69] COATS, J., "A Case of Simple Diffuse Goitre, with Secondary Tumours of the Same Structure in the Bones of the Skull", *Trans. Pathol. Soc. Lond.*, 1877, xxxviii, 399.
[70] HAECKEL, H., "Ueber eine Geschwulst von schilddrüsenartigem Bau im Unterkiefer", *Cor.-Blatt. d. allgem. ärztl. Vereins f. Thuringen*, 1889, xviii, 18.
[71] LITTEN, "Mitteilung über einen Fall von Carcinom der Thyreoidea", *Berl. klin. Woch.*, 1889, xxvi, 1094.
[72] FEURER, G., "Paradoxe Strumametastase", *Festschr. f. Kocher*, 1891, 273.
[73] EWALD, C., "Zwei Fälle von Kropfmetastase", *Wien. klin. Woch.*, 1893, vi, 459.
[74] KRASKE, Chir. Congress, 1893, and Boris-Bontsch-Ormdowsky Dissert., Freiburg, 1893.
[75] MIDDELDORPF, K., "Zur Kenntniss der Knochenmetastasen bei Schilddrüsentumoren", *Arch. f. klin. Chir.*, 1894, xlviii, 502.
[76] VON HOFFMAN, K., "Vier Fälle von Strumametastasen in Knochen", *Wien. klin. Woch.*, 1897, x, 1004.
[77] JAEGER, R., "Ueber Strumametastasen", *Beitr. z. klin. Chir.*, 1897, xix, 493.
[78] MUZIO, *Acad. d. med. d. Turin*, 1897.
[79] HONSELL, B., "Ueber gutartige metastasierende Strumen", *Beitr. z. klin. Chir.*, 1899, xxiv, 112.
[80] ODERFELD, H., and STEINHAUS, J., "Zur Casuistik der Knochenmetastasen von normalen Schilddrüsengewebe", *Zentralb. f. allgem. Pathol. u. pathol. Anat.*, 1901, xii, 209, and 1903, xiv, 84.
[81] HOLLIS, W. A., "Case of Paraplegia with Multiple Thyroid Tumour", *Lancet*, 1903, i, 884.
[82] DE GRAAG, K. S., "Ueber Strumen mit Knochenmetastasen", *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, xi, 625.
[83] PATEL, "Tumeurs bénignes du Corps thyroide donnant des Métastases", *Rev. de Chir.*, 1904, xxix, 398.
[84] HALBRON, P., "Cancer thyroidien developpé au Niveau du Sternum et simulant un Aneurisme de l'Aorte", *Bull. et Mém. Soc. Anat. de Paris*, 1904, lxxix, 373.
[85] DERCUM, F. X., "Thyroid Metastases in Spine", *Jour. Nerv. and Ment. Dis.*, 1906, xxxiii, 153.
[86] HALPÉRINE, *Thèse de Zurich*, 1906–7.
[87] BEILBY, G. E., "Affections of Thyroid Gland", *Albany Med. Ann.*, 1907, xxviii, 92.
[88] KOCHER, T., "Zur klinische Beurteilung der bosartigen Geschwülste der Schilddrüse", *Deut. Zeits. f. Chir.*, 1907, xci, 197.
[89] JABOULAY, "Goitre cliniquement bénin avec Métastase osseuse ayant simulé un Sarcome aneurysmatique de l'Extrémité supérieure de l'Humérus", *Lyon méd.*, 1908, cxi, 859.
[90] GUIBÉ, M., and LEGUEU, M. F., "Tumeur de la Clavicule d'origine thyroidienne", *Bull. et. Mém. Soc. de Chir. de Paris*, 1909, xxxv, 117.
[91] ALAMARTINE, H., and BONNET, P., "Un Cas de Goitre bénin métastatique", *Lyon Chir.*, 1911, v, 605.
[92] KNAPP, A., "Metastatic Thyroid Tumour in the Orbit", *Arch. of Ophthalmol.*, 1923, lii 18.

A DEPRESSED FRACTURE OVER THE ANGULAR GYRUS: CLINICAL AND RADIOLOGICAL LOCALIZATION.

By A. P. BERTWISTLE, Leeds.

Since localized brain lesions due to finite trauma in the vicinity of the left angular gyrus are not very common, the following case may be of interest. It occurred in a right-handed quarryman of average intellect, who sustained a compound fracture in this region and subsequently developed a mild aphasia. A method was devised rendering it possible to visualize, in radiographs, the relationship of the underlying gyri and sulci to the skull as a whole and to the parietal bone.

H. L., age 23, was struck on the head by a large stone. On admission six hours later he was quite conscious, but had no recollection of the interval between the accident and his being placed in the ambulance, nor could he remember the accident.

Operation.—Ether was administered and an extensive scalp-wound was cleaned. I exposed a depressed fracture of the parietal bone some two inches from the interparietal suture and one inch anterior to the lambdoid suture. The fracture was extensively comminuted, with hairs lying between the fragments. The pieces were removed individually, exposing the brain, all trace of dura having disappeared. The cerebrum was lacerated, and loose portions were swabbed away. The wound was closed without drainage in two layers, and 2000 units of antitetanic serum were given.

Clinical Localization.—On the day following operation the patient complained of periods during which he was unable to form words. He would break off in the middle of a sentence, unable to continue, although obviously trying. When given the word for which he was searching he expressed relief and repeated it. Two days afterwards he was more fully examined. His speech had improved, but he experienced difficulty in naming objects, showing paraphasia, e.g., he called a safety-pin 'single', and watch 'waa'. His power of reading was impaired; he was able to read verbs and nouns correctly, but manifested paralexia in the case of prepositions, adverbs, and conjunctions, with marked repetition of the word 'for'. He stated that he quite understood what he was reading, and knew that he was making mistakes. Four days later his writing was examined. He made one mistake in giving his address, calling Station 'Shadown', and pointed to the word as being incorrect. Power of copying printed matter both in type and in writing was good. Reading was better than before, and dictation moderately well accomplished. No motor paralysis was evident, but the right abdominal reflex and left knee-jerk were less easily elicited than those of the opposite sides.

Ten days afterwards his reading was almost normal, but still rather slovenly. The visual fields were normal. The wound had almost healed, and he had suffered from no headaches after the first few days.

The symptoms referring to the cortical lesion are: (1) Partial alexia; (2) Mild sensory aphasia; (3) Slight agraphia. They point to a lesion in the neighbourhood of the angular gyrus. The lesion cannot have been very deep, as hemianopia was absent. It is interesting to note that, although the extent of cortex involved was quite considerable, being $1\frac{1}{2} \times 1\frac{3}{4}$ in. in area, and although there was laceration of the brain, the symptoms were transitory in their nature. The clinical findings were confirmed radiologically as described below.

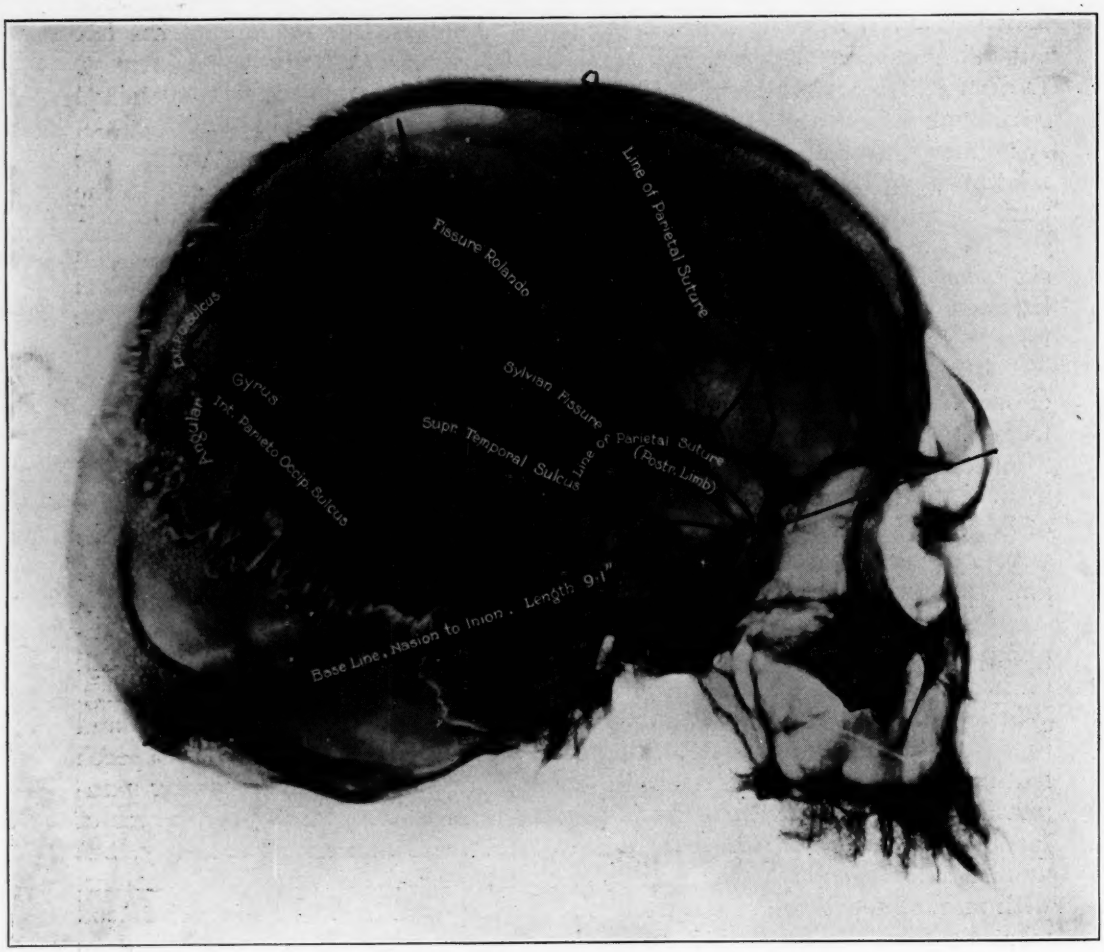

Line of Parietal Suture
Fissure Rolando
Sylvian Fissure
Line of Parietal Suture (Postr. Limb)
Supr. Temporal Sulcus
Int. Parieto Occip. Sulcus
Base Line, Nasion to Inion. Length 9.1"

FIG. 53.—Key radiogram, showing relations of brain to skull.

Radiological Localization.—The following method was devised to locate the site of the injury. The half of a sagittally divided skull with a corresponding half of a brain were taken. Between the more important gyri were implanted pieces of copper wire moulded according to the shape of the sulci. The boundaries of the parietal bone—where not obvious—were marked by glueing a thin copper wire on to its margin. The brain was placed in the skull and radiographed lying on its mesial surface, so that the picture is a

true lateral one (*Fig.* 53). It will be noticed that horizontal sulci lie nearer the vertex than is shown in text-books. The vertical ones show an apparent decrease in length which can only be accounted for by the convexity of the

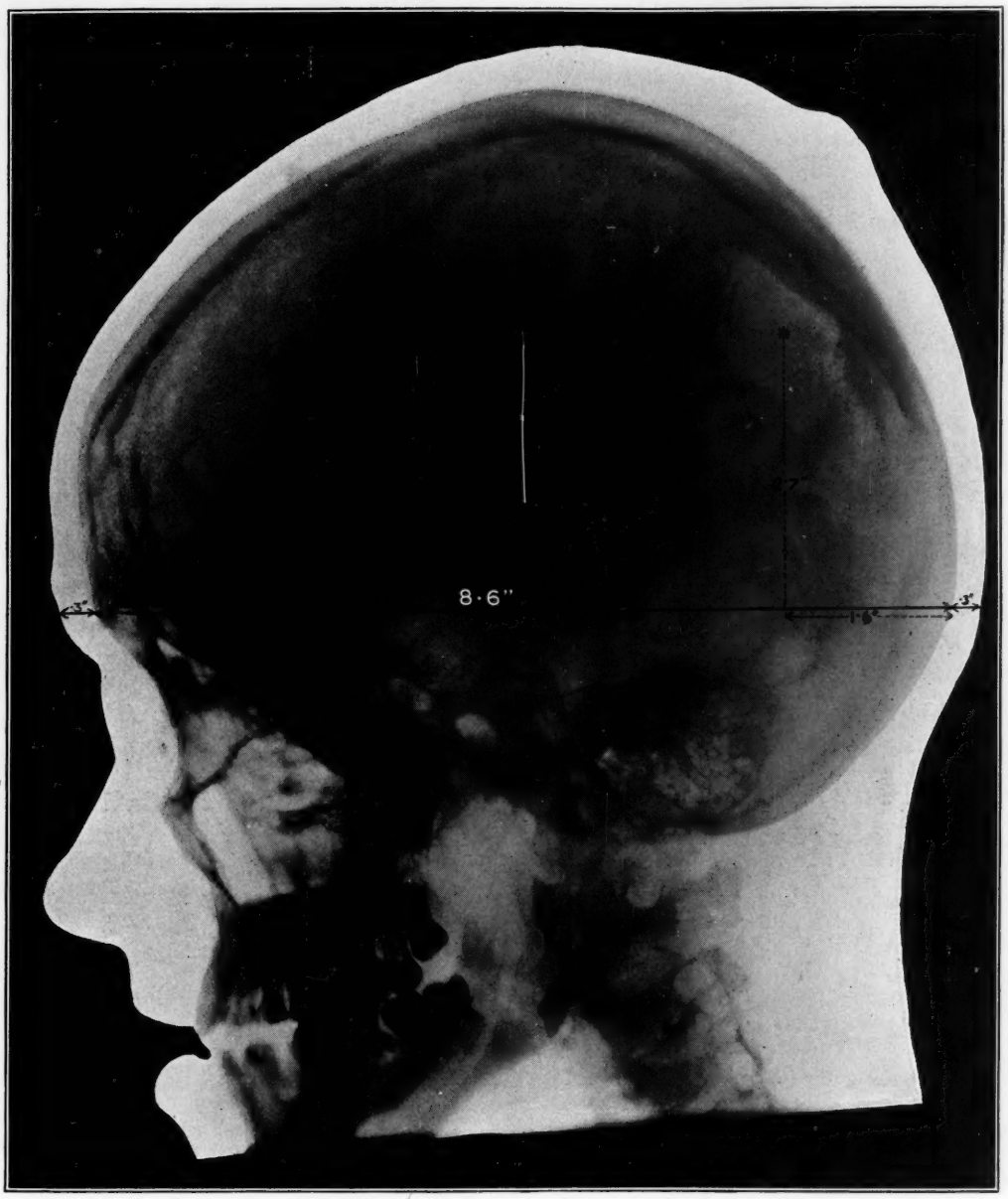


FIG. 54.—Depressed fracture over angular gyrus.

brain. This key radiograph is useful in reading radiographs of skulls, as one cannot compare the latter with the normal cranio-cerebral topographies of manuals; these depict the skull at different angles for the purpose of showing

the vertex. A base line is drawn from nasion to inion on both skiagrams, and by means of lengths along this line and perpendiculars to it localization is possible, as is now shown.

It is most necessary that the patient's skull should be in the same position as the key, i.e., purely lateral. This can be accomplished by placing a small lead bead in each external auditory meatus previous to radiography; the shadows cast by these spheres are then adjusted to lie one over the other. In this case the expedient was unnecessary in view of the almost complete superimposition of the inferior dental foramina. The base lines on skull and key measure 8·6 and 9·1 in. respectively, so that all horizontal distances before translation from skull to key must be multiplied by the constant $\frac{9{\cdot}1}{8{\cdot}6.}$ A perpendicular is now dropped from the middle of the gap in the skull to the base line (*Fig.* 54). It is found to be 1·6 in. from the posterior end, i.e., 1·6 in. $\times \frac{9{\cdot}1}{8{\cdot}6} = 1{\cdot}7$ in. from the inion of the key. The vertical distance between the base line and the vertex, at a point 1·7 in. from the inion, is 4·3 in. on the specimen and 4·5 in. on the key. The actual distance of the middle of the lesion from the base line is 2·7 in., so that the corresponding point on the key is $2{\cdot}7 \times \frac{4{\cdot}5}{4{\cdot}3} = 2{\cdot}85$ in. From these data it is obvious that the lesion lies over and behind the angular gyrus. It will be seen that the correction for size here is negligible, but in the case of a child it would be all-important.

SUMMARY.

1. A depressed fracture of the parietal bone was defined clinically as being over the angular gyrus by slight manifestations of sensory aphasia, alexia, and agraphia.

2. A key skiagram was prepared showing the relation of the skull to the convolutions of the brain.

3. By the use of base lines from inion to nasion and a constant, it is possible to localize, in radiograms, the area of brain involved in injuries of the skull, even though it be that of an infant.

My thanks are due to Mr. J. F. Dobson for permission to publish this case, and also to Dr. Moll for his kind help.

AN OPERATION FOR HÆMORRHOIDS.

By K. W. MONSARRAT, Liverpool.

I have employed the method of operating upon hæmorrhoids which is here illustrated for the last three years, and have found the results satisfactory.

Under general anæsthesia the anus is sufficiently dilated to bring the hæmorrhoids fully into view. If spinal anæsthesia is used this dilatation is unnecessary.

Three pairs of pressure forceps are applied to the hæmorrhoidal mass on one side of the middle line, and it is drawn over to the other side. A curved incision is now made through the skin just beyond the edge of the hæmorrhoidal semicircle (*Fig.* 55). The forceps are then applied to the inner edge of this wound. With touches of the knife and

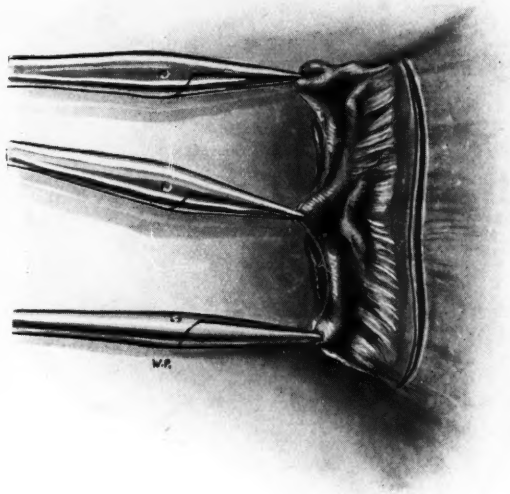


Fig. 55.—1*st step.* Pressure forceps applied. They should be shown divergent, putting the hæmorrhoidal mass on the stretch in the sagittal plane.

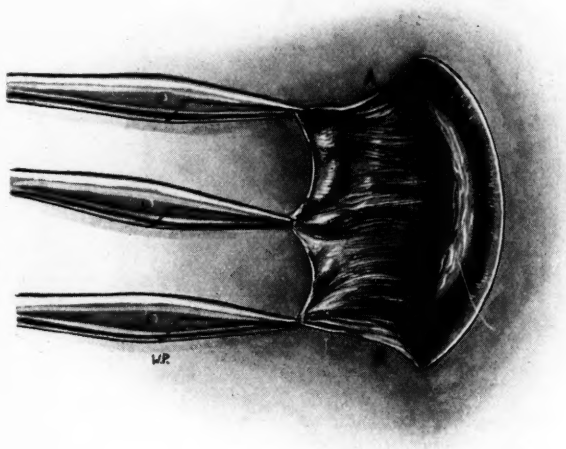


Fig. 56.—2*nd step.* Exposure of the external sphincter.

by gauze dissection the external sphincter is displayed; separation is continued between this muscle and the lining of the anal canal and rectum as far as may be judged necessary (*Fig.* 56). The degree of this denudation will vary with the degree of habitual prolapse. If there is much prolapse, small incisions are made with scissors at each pole of the semicircle (A and B, *Fig.* 56), but this is rarely necessary.

A clamp is then applied, and the mass cut so as to leave sufficient material beyond the clamp for suture (*Fig.* 57). The raw

edge so left is then sutured by a continuous catgut suture to the skin edge (*Fig.* 58). The clamp is removed and the suture pulled taut and tied; it is hæmostatic. The semicircle on the opposite side is treated in a similar way (*Fig.* 59). The wound is lightly wiped with B.I.P.P.

FIG. 57.—*3rd step.* Half of the mass cut away.

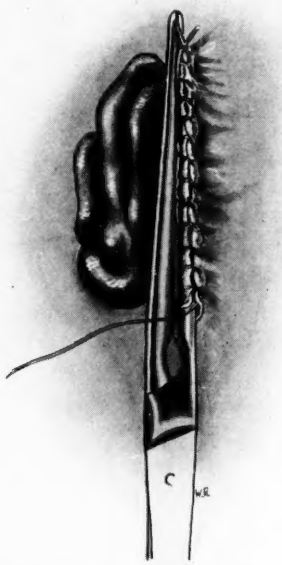


FIG. 58.—*4th step.* Raw edge sutured to skin edge.

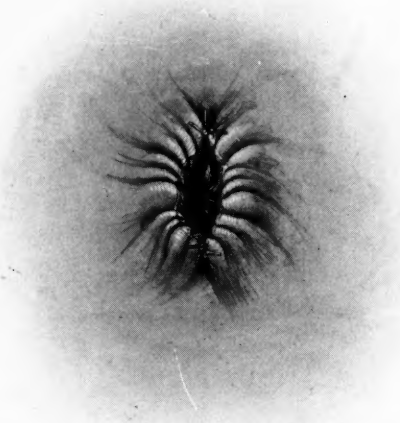


FIG. 59.—The operation completed.

The operation deals radically with the varices and also with any prolapse that may be present. Occasionally one or two arterioles are severed by the primary incision and need ligature; usually there is no hæmorrhage at all. The external sphincter comes fully into view and cannot be injured.

RETROGRADE INTUSSUSCEPTION OF THE SMALL INTESTINE AFTER GASTRO-ENTEROSTOMY.

By HAMILTON DRUMMOND, Newcastle-upon-Tyne.

During the last few years, cases of retrograde intussusception of the jejunum following gastro-enterostomy have been recorded, and recently one such came under my care. As this is a rare, but now a well recognized, complication of this operation, I think a record of my own case, with abstracts and observations from others, may be of interest. Hitherto eleven cases have been recorded, two of them in this country—one by Richard Warren, another by St. Bartholomew's Hospital—and the remainder on the Continent. My own case will make twelve in all.

The chief interest in the condition lies in the difficulty of explaining the factors which are responsible for the occurrence of this retrograde intussusception; why it should occur in a few cases only; and also what steps ought to be taken to prevent relapse, as in the case recorded by Baumann.

AUTHOR'S CASE.

R. H. B., age 35, a platelayer by trade, was admitted to the Royal Victoria Infirmary on July 25, 1922, complaining of severe spasmodic pain in the abdomen. He stated that he was well up to 10 a.m. on the previous day, when quite suddenly after taking food he experienced severe pain in the epigastrium. The worst pain came on in spasms, but it had also been continuous up to the time of his admission. He vomited freely after the pain commenced, and frequently during the day and night up to the time he was seen in the Royal Infirmary. At first the vomit consisted only of food which he had taken, but later it contained blood, and the specimen seen, which he brought up after being in hospital one hour, contained dark-coloured liquid blood and was foul smelling. He had passed no flatus, and his bowels had not been moved since the previous day, just before the pain came on.

On examination, he looked ill. His temperature was 98° and his pulse 96. His tongue was dry and furred. There was a mid-line incision the result of a previous operation above the umbilicus. The abdomen moved well on respiration, and no mass was palpable, nor was there any definite amount of tenderness or rigidity. Rectal examination was negative.

During the short time he was in hospital before operation he had several attacks of pain accompanied by vomiting of blood.

Previous History.—This was kindly given to me by Professor Francis Caird, of Edinburgh, who had operated upon him on two previous occasions for chronic duodenal ulcer, the first time sixteen years ago.

"March 3, 1906.—One year ago he complained of sudden pain in the stomach and swelling of the abdomen, not affected by taking food. It was so severe that he

took to bed, and after resting, with relief, the symptoms returned and he was obliged to lie up again. Now he began to vomit brown frothy sour-smelling fluid—no relation to food. He has been off work for a year, and for the past nine months has never been free from pain. He is always constipated, but has never vomited blood. No melæna. Test meal showed hydrochloric acid abundant. Stomach holds 3¾ pints. No peristalsis seen. Stomach when full descends to about one inch above the pubis.

"*Operation, March* 6.—Through a mid-line incision. Stomach lay completely above the umbilicus. Firm adhesions between the anterior surface close to the lesser curvature and the anterior abdominal wall. The omentum was adherent to the abdominal wall also. The stomach was large and hypertrophied. The pyloric end was firmly buried in adhesions, which also involved the duodenum. The adhesions were divided with scissors, exposing a very thickened and stenosed pylorus. All the coils of small intestine lay on the right side of abdomen below the liver, to the right of the ascending colon and hepatic flexure. The cæcum and appendix lay far to the left of the mid-line. To get a suitable loop of jejunum for gastro-enterostomy, the adhesions covering the duodenum had to be lacerated with a blunt dissector. It was considered advisable to perform a Roux Y operation. The opening in the mesentery of the jejunum was stitched up and also united with another portion of mesentery to avoid the probability of an internal strangulation.

"He made a good recovery, and on March 19 was on full diet. On March 22 he left for the Convalescent Home.

"He was re-admitted on April 20, 1909, complaining of pain twenty minutes after food, associated with vomiting. Since leaving hospital after the operation in 1906, although he did not put on much weight, he was in the best of health until December, 1908. Then sharp pains were experienced in the gastric region twenty minutes after a meal. They did not enter into his back. He had no vomiting, nor had he melæna. At times frequent heartburn and waterbrash, accompanied by flatulence, troubled him very much. His bowels became irregular and failed to act daily. The pain steadily became more severe, lasting on an average for three or four hours. In February, 1909, he began to vomit at odd times about once every three weeks, three or four hours after a meal; sometimes he would vomit during the night. The vomit at that time was brownish and frothy. During the last three months he has lost fourteen pounds in weight. The stomach stands out in rigid spasms at times, peristaltic waves passing from left to right. Marked splashing of the lower border of stomach at umbilicus. There was no tumour palpable; no tenderness; test meal showed stomach to contain plenty of hydrochloric acid.

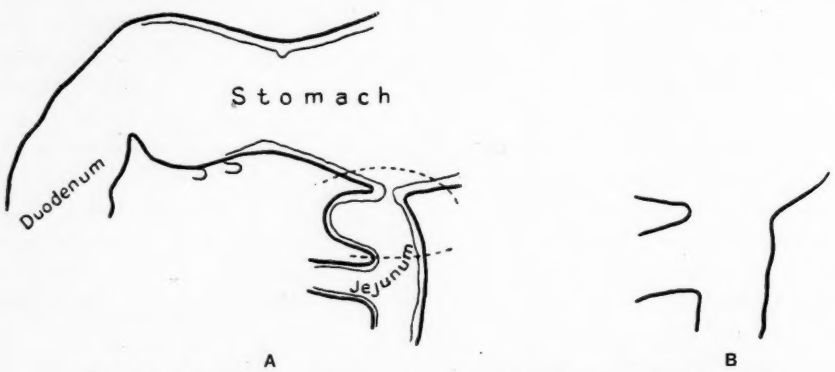


FIG. 60.—Removal of gastro-enterostomy opening and pouch below. (A) The dotted lines show the area resected. (B) After resection and suture.

"*Second Operation.*—He was kept under observation for some time, and on May 3, 1909, was operated upon for the second time. On opening the abdomen the relations to the large and small intestine were not so abnormal as at the former

operation. The gastric stoma was very much narrowed. The pylorus was larger than when previously noted. There were several enlarged glands, one of which was removed for section. The stoma and adjacent portion of the jejunum were removed, and a fresh union above the Y to the stomach was made. From the diagram (*Fig.* 60) it will be seen that the portion of jejunum close to the stomach had become pouched. This was removed."

The patient left for the Convalescent Hospital on May 25, 1909, feeling very well, and he remained well until his present illness.

Present Illness.—

Third Operation, July 25, 1922, *two hours after admission.*—On opening the abdomen through the old scar, 35 hours after the onset of acute pain, very few adhesions were found between the viscera and the abdominal wall. Posterior gastro-enterostomy had been done with a long loop. The proximal loop was enormously distended, being three or four times its normal size. This dilatation commenced at the first part of the duodenum, and ended at the junction of the jejunum with the stoma ; the portion of stomach between the stoma and pylorus was small and atrophied, and was covered in by old fibrous adhesions. The distal side of the bowel below the anastomosis for about six feet had become intussuscepted into the stomach through the stoma. The intussuscepted gut was easily reduced. It was very œdematous, and showed areas of hæmorrhage into its walls, having in every way a similar appearance to the intestine found in cases of ileocolic intussusception. My first impression was that the gut was being reduced from the lesser sac, but this was found not to be the case, and the lesser sac was connected with the greater by a large opening in the mesocolon. The stoma was situated almost on the greater curvature of the stomach, and admitted two finger-tips when palpated through the jejunal wall. Owing to the man's shocked condition it was decided to do nothing more after reduction of the intussusception.

Fourth Operation, Aug. 10, 1922.—He made a straightforward recovery from the last operation, and his wound having healed by first intention, and his temperature and pulse being normal, his abdomen was again opened with a view to attempting to prevent further recurrence of the retrograde intussusception of the small intestine into the stomach. Again the mid-line incision was used, and a new anastomosis was made between the stomach and the jejunum, after separation and suture of the old one. A proximal portion of the jejunum was taken to the old site of anastomosis, leaving no loop. The duodenum and the upper part of the jejunum were again noticed to be very distended and hypertrophied. (The appendix and cæcum were in the left side of the abdomen, high up in the position of the spleen, as noted by Professor Caird at his first operation.)

The patient made a straightforward recovery, and went home on Aug. 14. He was seen from time to time in the out-patients' department, and continued to do well.

On Nov. 1 he was again re-admitted to the hospital, with the following history : Since he returned home on Aug. 14 he had been in excellent health until the previous evening (twenty-four hours previous to admission), when, whilst playing cards in the house, he was suddenly seized with very severe pain in the epigastrium which, he states, ' dropped ' him. He was then

just able to get upstairs without assistance. After getting there he fainted, was put to bed, and the doctor was sent for. He did not sleep at all during the night, and was unable to take any food, but had not vomited up to the time of his admission to hospital next morning. The bowels had been moved on the morning before his attack at 10 a.m.

On examination, after being in hospital for an hour, he was found lying on his left side. He stated that he felt a little better, but the pain, which was continuous, and not in spasms as it had been in his previous attack, was still present. He did not look so ill as he had done on the last occasion. His pulse was 128 and his temperature 99·8°. His tongue was clean. When asked to turn on his back, he did so, but only slowly and with difficulty. His abdomen was distended in the upper part, and respiratory movements were almost absent. He was tender on deep pressure on the right side, and there was marked rigidity of both recti muscles above the umbilicus. Liver dullness was diminished but present. He was dull in both flanks, more marked on the left side. No mass was palpable.

Fifth Operation, Nov. 1, 1922.—A diagnosis of perforated viscus was made, possibly a jejunal ulcer, and he was operated on at once, twenty-five hours after the onset of pain. The old mid-line incision above the umbilicus, which had healed well, was opened up. There was purulent fluid in the abdomen among the coils of small intestine, which were distended and intensely injected. On exploration of the stomach in the gastro-enterostomy region, which was shut off by plastic lymph, gas escaped from a small hole the size of a goose quill. This was found to be in the loop of jejunum just beyond the gastro-jejunostomy opening which had been made two months before. The loop of proximal jejunum and the duodenum were still dilated, but this was nothing like so marked a feature as had been noted on the previous occasion. The ulcer was closed by a purse-string suture of catgut followed by three interrupted Lembert sutures of silk. Considerable thickening round the ulcer appeared to be due to jejunum œdema, the result of recent inflammation. The abdomen was mopped out with saline mops, and it was found that fluid was present as far down as the pelvis. The cæcum on this occasion was noted to be in its normal position, but with a very long mesentery. The abdomen was closed by sutures in layers.

The patient made a straightforward recovery. The wound healed well, and on Nov. 4 he was eating chicken. He left hospital on Dec. 8 perfectly healed and on ordinary diet, with directions to take for at least a year regular large doses of the mixed alkalis, carbonate of soda, magnesia, and bismuth.

CASES RECORDED IN THE LITERATURE.

Lundberg[1] describes a case of his own of retrograde intussusception following gastro-enterostomy, and records eight other cases of a similar kind.

His patient was a woman, age 48, who had been operated on in 1911, and at that time was supposed to have carcinoma of the stomach. The pyloric end of the stomach was resected and an anterior gastro-enterostomy with entero-anastomosis performed. The stomach was very large, and there was a definite tumour at the pylorus. She had remained well up to twenty-four hours before she came under his care in 1921. She then complained of pain in the upper abdomen, and vomiting, and was unable to

account for this by any error in diet. The vomited matter first contained bile and afterwards blood. On admission to hospital her temperature was normal; pulse feeble, 100. She had pain and repeated vomiting attacks, the vomited matter containing blood. The abdomen was not distended. A tumour could be distinctly felt under the left lower ribs, about two fingers in breadth. It was hard and mobile. The patient grew worse in spite of stimulants and saline transfusion. On the following day the vomiting of blood ceased; but she grew worse, and died four days after her initial attack. There was no operation in this case on account of the patient's bad general condition.

Post-mortem showed that the distal loop of jejunum was intussuscepted in a retrograde direction upwards into the stomach. It entered the anastomosis through the distal loop of jejunum to the gastro-enterostomy opening. The intussuscepted bowel in the stomach was twenty inches in length. It was much inflamed. The stomach and intussuscepted intestinal loops were filled with fluid blood. There was no peritonitis, and no growth present.

Other cases quoted by Lundberg may be briefly mentioned:—

Steber's case:[2]—

A woman, age 21, was operated upon by Steber for stomach trouble. Posterior gastro-enterostomy was done. She was seven months pregnant, and had suffered from vomiting the whole of this time. She was suddenly seized with severe pains in the stomach with violent vomiting, and four hours later blood was noticed in the vomit. A diagnosis of bleeding ulcer was made. The cramplike pains in the epigastrium continued, but there was no rigidity. She died on the third day after the initial symptoms.

Post-mortem examination showed an intussusception of the distal jejunal loop of the jejunum into the stomach 30 cm. in length. The base of the intussusception appeared to be at the gastro-jejunostomy opening.

Baumann's case:[3]—

A woman, age 44, had been operated on for ulcer of the duodenum. The pylorus was occluded and anterior gastro-enterostomy was performed with entero-anastomosis. An intussusception into the stomach was reduced, and the patient did well. Eight weeks later she had a return of the same cramping pains, and twenty-four hours after the commencement of her second attack a tumour could again be felt, which was lying a hand-breadth below the umbilicus on the left side. She vomited 1½ litres of foul-smelling liquid, and after this the tumour was noticed to be at a higher level. The condition was diagnosed (retrograde intussusception), and the abdomen was opened again. The base of the intussusception was now lying about 25 cm. below the entero-anastomosis, and the apex reached to the gastro-enterostomy opening. It was not possible to reduce the intussusception completely, and about 10 cm. of intussuscepted gut was resected.

The patient was operated upon later for intestinal strangulation, after which she recovered.

Hatert's case:[4]—

A man, age 30, was operated on for perforated ulcer of the stomach, which was closed by suture and a posterior gastro-enterostomy performed. Nine months later he was taken ill during the night with severe pains in the stomach and vomiting. On the following morning the vomiting continued and he brought up blood. The stomach was distended, but the remainder of the abdomen was normal. Under the left rib margin was a tumour the size of a fist. Operation was performed, a diagnosis having been made of obstruction in the jejunum. It was found that a loop of jejunum was intussuscepted into the stomach for 30 cm. The base of the intussusception was thought to be at the margin of the gastro-enterostomy opening.

Reduction was performed and the patient recovered.

Schloessmann's case :[5]—

A woman, age 42, had been operated upon ten years previously for a growth of the stomach, and gastro-enterostomy had been performed. She was ill for two days, and complained of severe sudden pain in the abdomen. Half an hour later vomiting began which continued day and night, accompanied by repeated cramps in the stomach. At first the vomit contained bile, and in twenty-four hours blood was noticed, which became more marked later on. The abdomen was not distended or rigid, though the recti in the region of the epigastrium were contracted and tender. The remainder of the abdomen was soft, and there was no distention. At the level of the umbilicus, lying horizontally, there was a swelling about the size of a fist which was tender on pressure. A diagnosis of intestinal obstruction high up in the small intestine was made. At the operation 40 cm. of the distal jejunal loop was found intussuscepted into the stomach through the gastro-enterostomy opening. The intussusception was reduced. In order to prevent this happening again the distal loop of jejunum was sutured to the colon.

An uneventful recovery followed, though three weeks after operation the patient complained of severe short cramping pains.

Amberger's case :[6]—

A man, age 43, five years previously had had anterior gastro-enterostomy performed with entero-anastomosis. There was marked pyloric obstruction. The patient for several years was free from pain, and then began to have severe vomiting, which became fæculent, and he was unable to get his bowels moved or to pass flatus. He was in very bad condition. At the operation 12 cm. of the distal loop of jejunum was found to be intussuscepted upwards, with the apex of the intussusception close to the entero-anastomosis.

Resection was undertaken and the patient recovered.

Lundberg also mentions two cases which came under the care of Eberle,[7] where the distal jejunum loop became the seat of an ascending intussusception. In these cases anterior gastro-enterostomy had been performed. They were operated upon and both recovered.

The following case came under the care of Mr. Richard Warren,[8] of London :—

E. S., a male, was admitted into the London Hospital suffering from hæmatemesis. Thirteen years previously gastro-jejunostomy had been performed at another hospital for some stomach lesion. The patient made a good recovery, and remained well for twelve years.

One year before admission he began to suffer from abdominal pain after meals, accompanied by vomiting. The sickness relieved the pain. At times he would be free from stomach trouble for a period extending over a month. Two days before admission he had a very bad attack of pain associated with hæmatemesis, which continued up to the time of his admission. On examination he was found to be very ill, and showed the abdominal facies. The pulse was 100. There was considerable vomiting of large quantities of blood-stained fluid. The abdomen was flaccid, and an indefinite elongated mass was palpable above and to the left of the umbilicus. On account of the softness of the mass, the suggestion that it was due to a peptic jejunal ulcer was dismissed.

At the operation a few adhesions were found binding the transverse colon to the abdominal wall. These were separated. The stomach and pylorus appeared to be normal. On turning up the stomach to examine the stoma the distal loop of the jejunum was found to be turgid and purple for 8 inches, where the entrance of a retrograde intussusception was found. The intussusception passed up the distal jejunal limb, its apex projecting about 3 inches into the stomach through the stoma. The reduced gut was very œdematous and purple, but at no point could any thickening suggestive of ulceration be felt. The abdomen was closed.

The patient vomited some brown fluid on the day following the operation, but continued to improve until the seventh day, when respirations became rapid and he had foul offensive sputum. Death occurred on the tenth day.

Post-mortem showed the lungs to be in a condition of severe bronchial pneumonia with multiple abscesses. The abdomen was healthy except for slight plastic peritonitis about the site of the intussusception and some bruising of the intestine. The gastro-enterostomy as seen at the operation proved to be in excellent order. The stoma was of good size and well placed. There was no sign of any peptic ulcer in the jejunum.

Case reported by Dr. Arnsperger[9] :—

A woman, age 59, had posterior gastro-enterostomy performed for pyloric stenosis. Eight days later she had an entero-anastomosis done owing to symptoms of vicious circle. She remained fairly well after this until eleven and a half years later, when she was suddenly seized with colicky pains in the stomach, with coffee-ground-like vomiting. On the following day (twenty-four hours after onset of pain) her general condition was good, pulse was 100, the abdomen was flaccid, and in the left hypochondrium on deep palpation there was definite resistance found.

Operation was performed twenty-four hours after onset of pain, and stomach and omentum were found to be adherent to the anterior abdominal wall. After freeing and retracting the stomach and colon a sausage-shaped intussusception was found. The first part of the small intestine was very distended. The intussusception lay 20 cm. below the gastro-enterostomy. The apex was close to the entero-anastomosis. Reduction was accomplished with some trouble by pulling and squeezing the intestine. The entero-anastomosis appeared to be very wide, and the gastro-enterostomy was acting well. The abdomen was closed in layers without drain, and recovery was straightforward. One month after operation the stomach and bowels were acting perfectly well and the general health was excellent.

The patient later stated that during the first year after her first operation she had three attacks of stomach colic but without any vomiting. They were similar to the present attack, and had been cured by massage. These attacks lasted from two to three hours. She was unable to account for the attacks.

ETIOLOGY.

The condition known as retrograde intussusception has been recognized for a long time, and occurs apart from gastro-enterostomy. Leichtenstern,[10] after careful examination of the literature on this subject, found that out of 593 cases of intussusception, only 8 were of the ascending type. According to Baumann's statistics, one ascending intussusception occurs to 200 descending cases.

There can be no doubt that, in the cases to which I wish to draw attention, the gastro-jejunostomy is in some way the primary cause of the ascending intussusception. There is one thing quite certain, and it is that whatever the cause of the retrograde intussusception be, it is not due to any misplacement of the stoma between the stomach and the intestine. It does not appear to matter which type of gastro-jejunostomy has been performed. Of the twelve cases in which intussusception occurred, five were anterior gastro-enterostomies, five posterior, and in the remaining case no note was made.

In some of the cases an entero-anastomosis had been performed in addition to the gastro-enterostomy; in fact, it is a point of special interest to note that this had been done in five of the twelve cases.

Retrograde intussusception has also been recorded to occur above a stricture of the small intestine, as if attempts on the part of the bowel to

empty itself by forcible antiperistalsis had been made. Riedel[11] records a case of ascending intussusception of the colon some distance above a descending intussusception.

The most reasonable cause of ascending intussusception in these cases appears to be as follows: In the first place, there must often be a tendency in cases of gastro-enterostomy for the stomach to empty itself through the stoma more rapidly than is normal, its acid contents causing some irritation of the upper jejunum, as was shown by the development of a ruptured peptic jejunal ulcer in my case. This fullness and acidity of the contents of the upper jejunum might result in forcible antiperistaltic action, the contents being regurgitated into the stomach through the stoma, which, unlike the pylorus, has no power of preventing this, as it is possessed of no sphincter action. Then, when the intestine enters the stomach, further portions of it are drawn in by the efforts of the stomach to eject its contents.

SYMPTOMS AND DIAGNOSIS.

Having once seen a case of ascending intussusception following on gastro-enterostomy, the symptoms are definite enough to suggest a diagnosis of the condition. It appears that Baumann made an accurate diagnosis in his case, as the same condition recurred eight weeks later. These symptoms following gastro-enterostomy are: sudden attacks of cramping pain in the epigastrium, followed at first by vomit of food and bile, and later of blood; inability to pass flatus and to have the bowels moved. Rigidity and distention do not appear to be often present, nor is tenderness constant. In six of the known cases a tumour was noticed. In all cases, so far as is known, where the intussuscepted intestine reached the stomach, blood in the vomit was a marked feature. By this time the strangulation and interference with the blood-supply would be acute and explain this symptom. Though the facial aspect denotes a serious illness, rise in temperature does not seem to be a prominent feature.

It is a noteworthy fact that in the majority of cases intussusception occurred a long while after gastro-enterostomy had been performed. In nine of the twelve cases recorded, where definite dates are given, the invagination occurred 15½ years, 12 years, 11½ years, 10 years, 10 years, 5 years, 5 years, 1½ years, and nine months after the operation of gastro-enterostomy, making the average nearly 7 years after the first operation.

In two of these the patients were pregnant. One, a woman 21 years of age, was seven months gravid (Steber's case), and had suffered from the commencement of her pregnancy from persistent vomiting, when suddenly this, accompanied by pain, became very much worse and she vomited blood four hours later. The symptoms continued until she died on the third day. Post-mortem showed 30 cm. of the distal limb of the jejunum to be lying in the stomach.

In the second case, for the notes of which I am indebted to the authorities of St. Bartholomew's Hospital Museum, a woman was admitted to hospital for persistent vomiting in the thirty-fourth week of her second pregnancy. She died three days later, and post-mortem showed 2 feet of distal loop of

jejunum intussuscepted into the stomach through the stoma (*Fig.* 61). Gastro-enterostomy in this case had been performed five years previously.

In these two cases it would seem that persistent vomiting, the result of the pregnancy, may have played a part in producing the intussusception.

FIG. 61.—Retrograde intussusception of small intestine into the stomach through the stoma of a posterior gastro-enterostomy which had been performed five years previously. Two feet of intussuscepted jejunum are seen lying in the interior of the stomach. The pyloric opening and duodenum are seen on the left-hand side of the illustration.

(*From a specimen kindly lent by St. Bartholomew's Hospital Museum.*)

TREATMENT.

The treatment of this condition is certainly, as in all forms of intussusception, operation. It is difficult to know what could assist in preventing the condition from occurring a second time, as in the case recorded by Baumann. Schloessmann in his case sutured the involved loop of jejunum to the colon, after reduction of the intussusception. In my own case, partly on the ground of enormous distention of the proximal loop of jejunum, and to prevent perhaps a further recurrence, a second operation was performed ten days later, when a fresh stoma was made in the stomach, attaching the loop of jejunum higher up the bowel, making a short-loop junction. This, unfortunately, did not prevent the patient from returning to hospital eleven weeks later with an acute ruptured peptic jejunal ulcer, as recorded in the notes.

Since this paper was completed, two more cases have come under the author's notice, reported by Lewisohn[13] and Delfino.[14] In both cases the intussusception occurred a short while after gastro-enterostomy had been performed.

CONCLUSIONS.

1. Retrograde intussusception of the small intestine is now a well-recognized complication following gastro-enterostomy. Fourteen cases have been recorded.

2. The type of gastro-enterostomy performed has nothing to do with the occurrence of the ascending intussusception.

3. In all probability the ascending intussusception is caused by too rapid emptying of the stomach, causing irritation of the jejunum, and setting up forcible antiperistalsis.

4. Diagnosis is straightforward, and should, now that the condition has been recognized, be easily made.

5. The history of a previously performed gastro-enterostomy, often of many years' standing, followed by sudden griping epigastric colic, vomiting of blood, often a palpable tumour in the left hypochondriac region, with absence of rigidity, distention, and acute tenderness, suggest the diagnosis.

6. The treatment of the condition is immediate operation.

7. A reliable preventive treatment has not been suggested.

REFERENCES.

[1] LUNDBERG, S., *Acta Chir. Scand.*, 1922, No. 54, 423.

[2] STEBER, "Ueber einen seltenen Fall von Invaginations-Ileus", *Münch. med. Woch.*, 1917, No. 20, 648.

[3] BAUMANN, "Wiederholte aufsteigende Dünndarminvagination bei einer gastro-enterostomierten Frau", *Arch. f. klin. Chir.*, cxi, 504; "Aufsteigende Jejunum-invagination nach Gastroenterostomie, *Zentralb. f. Chir.*, 1921, No. 42, 1543.

[4] HATERT, "Dünndarminvagination in den Magen nach Gastroenterostomie", *Ibid.*, 1921, No. 32, 1154.

[5] SCHLOESSMANN, "Rückläufige Dünndarminvagination in den Magen nach Gastro-enterostomie", *Ibid.*, No. 42, 1538.

[6] AMBERGER, "Dünndarminvagination in den abführenden Schenkel einer vorderen Gastroenterostomie", *Ibid.*, 1541.

[7] EBERLE, "Zu den Komplikationen der Gastroenterostomia retrocolica", *Ibid.*, No. 29, 1044.

[8] WARREN, R., *Lancet*, 1919, ii, 615.

[9] ARNSPERGER, L., "Retrograde Dünndarminvagination nach Gastroenterostomie", *Zentralb. f. Chir.*, 1922, No. 49, 190.

[10] LEICHTENSTERN, quoted by *Nothnagel's Encyclopedia of Practical Medicine*, "Diseases of Intestine and Peritoneum", 499.

[11] RIEDEL, quoted by LUNDBERG, *Acta Chir. Scand.*, 1922, No. 54, 423.

[12] *St. Bartholomew's Hospital Museum Catalogue*, London.

[13] LEWISOHN, R., "Intussusception of Stoma after Gastro-enterostomy", *Ann. of Surg.*, 1922, lxxvi, 543.

[14] DELFINO, E., "A rare complication of Gastro-enterostomy", *Deut. Zeits. f. Chir.*, 1922, clxxiv, 69.

CHRONIC MASTITIS.

By GEOFFREY KEYNES, London.

(Being the Hunterian Lecture delivered at the Royal College of Surgeons of England on May 9, 1923.)

THE PATHOLOGICAL PROBLEM PRESENTED BY MASTITIS.

The pathological problem underlying the clinical condition known as chronic mastitis is one that is ever present to those engaged in the practice of surgery; yet the current ideas concerning it are usually vague, and often, I believe, erroneous. It is known to be an exceedingly common condition, though only those patients present themselves for treatment who suffer from the extremer degrees of it, so that it is certainly even commoner than ordinary clinical experience would suggest. Many women through long habituation come to regard a certain degree of pain in their breasts as a normal state of affairs, and consequently never seek advice concerning it. To those who do come to the surgeon the treatment that is meted out is often unsatisfactory, for the local application of a belladonna plaster, and advice as to wearing a support, can only be regarded as palliatives of doubtful value.

The condition is most often seen in women nearing the menopause, that is to say, between the ages of 40 and 50 years. The patient complains of an aching or dragging sensation in one or both breasts. Sometimes she will state that there are pricking pains at different points in the breast, and nearly always there is an increase in the symptoms at the menstrual periods. Sometimes she will state that there is an intermittent discharge of fluid from the nipple, but usually this is not noticed.

On palpation the breast is found to be tender, especially at certain points, and if the patient be stout this is often all that can be made out. If the patient be thin the breast substance is found to have a characteristic ropy feeling, and the gland may be diffusely enlarged. Usually no definite tumour can be felt, but the breast substance is found to be studded with small raised points, and some of these knobs may be the centres of tender spots. Often the axillary lymph glands are also found to be somewhat enlarged and tender, though they remain soft in consistency, unlike the lymph glands associated with carcinoma.

The pain, tenderness, and glandular enlargement all suggest that the breast is the seat of an inflammatory lesion, and this interpretation is implied by the name that is given to it. Surgical teaching is, however, commonly somewhat reticent with regard to the further details of the condition and to its etiology. The inquiring student is given to understand that there are three types of mastitis: (1) *Chronic interstitial*, or *lobular*; (2) *Chronic lobar*; and (3) *Chronic parenchymatous mastitis*; and, knowing that most inflammations are due to the action of pyogenic bacteria, he assumes that some 'subacute' infection is here responsible. He has probably also seen cases of

abscess in the breast following lactation, and knows that then there is a definite infection, usually with the *S. pyogenes aureus*. The two conditions thus become connected in his mind, and he perhaps imagines that the chronic condition is sometimes an aftermath of the acute. He is thus confirmed in the bacterial interpretation of chronic mastitis, though he finds later that this knowledge gives him but little help in treating the disease.

Afterwards, as clinical experience accumulates, the former student whose mental processes we are examining notices that there is in fact no connection between chronic and acute mastitis. The suppurative lesion is almost restricted to the period of lactation, whereas the chronic condition occurs at all ages, and is perhaps commonest in women who have never borne children. Certainly an acute mastitis cannot be shown to be a predisposing cause of chronic mastitis. Clinical observation still affords no help in distinguishing between the interstitial and parenchymatous varieties, nor are any of the ordinary clinical signs of bacterial infection to be found. The condition is not one which is dangerous to life, and usually does not amount to more than discomfort in the sufferer. A woman who complains that her life is made miserable by it is apt to be classed as neurasthenic, and palliative treatment is given to the others.

But if chronic mastitis be not bacterial in origin, what is it? If it is an insidious change taking place independently of any acute infection in the breast, when and where are its obscure beginnings? If the terms 'interstitial' and 'parenchymatous' indicate merely academic subdivisions of one condition, what then are the exact changes to be seen under the microscope, and what is their relative importance? Is chronic mastitis really a clinical entity at all, and if not, what is its relation to other diseased conditions of the breast? Lastly, if a true understanding of the condition is not of great intrinsic importance, may it perhaps have some bearing on the vastly important question of the cause and prevention of cancer of the breast? These are the pathological problems that present themselves.

THE PLAN OF THE PRESENT INVESTIGATION.

The investigation of chronic mastitis which I have recently attempted was undertaken at the suggestion of Professor G. E. Gask, and was carried out in the laboratories attached to the Surgical Professorial Unit at St. Bartholomew's Hospital. It was proposed that a mammary gland should be removed from every female patient that came into the post-mortem room at the hospital during a given period, and that it should be submitted to a careful macroscopic and microscopic examination. It was hoped that some information would thus be obtained concerning the early stages of pathological changes in the breast and as to the average age at which they become apparent. It was felt, too, that the investigation would also help to establish, at any rate in our own minds, a clear idea of the detailed histology of the condition. The material obtained from this source has provided the bulk of the results which are recorded here.

At the same time I have tried to investigate three other series of specimens: firstly, some male mammary glands collected at random in the

post-mortem room; secondly, material removed from female patients operated upon for the grosser degrees of clinical chronic mastitis; thirdly, mammary glands removed for carcinoma.

Clearly the post-mortem room material would afford no cultural information as to the bacterial origin of the disease, but would be entirely histological in its bearings. On the other hand, the material obtained at operation would afford histological evidence of the later stages of the disease and could be tested bacteriologically.

The pathological material thus obtained has been hardened in 10 per cent formalin. Each breast has then been cut into serial slices, carefully examined with the naked eye, and portions have been selected for microscopic examination. I am aware that at the present time it is considered in some quarters that the only satisfactory way of examining a breast is by means of the large-scale or 'window-pane' sections of the whole gland, and the method I have used has been somewhat contemptuously designated 'the cheese-tasting method'. I agree that large-scale sections would be the ideal, but the technical difficulty of making large numbers of these preparations has deterred me, and the expense of the apparatus that is needed has proved prohibitive. I would also submit that sections of a relatively small size give more accurate histological detail than the best of sections made on the more grandiose plan, but I stand open to correction on this point.

Lastly, I have had under observation as out-patients a certain number of women in whom chronic mastitis had been diagnosed, but not of a degree requiring operation.

THE HISTOLOGICAL STRUCTURE OF THE NORMAL BREAST.

At the outset of any investigation of this kind it is obviously necessary to have a clear conception of the histological structure of the normal organ, but in the case of the mammary gland this is not so simple a matter as might be supposed. Changes, presumably pathological, proved to be so common that an absolutely normal adult organ was difficult to find. Also the gland is subjected to so many violent physiological changes that a great many different aspects are regarded as normal. An attempt, however, will be made briefly to describe the normal with regard to the life-history of the individual.

The mammary gland, developed early in fœtal life from modified sweat glands, is at birth much the same in either sex. Of true glandular tissue there is little or none, the organ consisting of simple ducts, between twelve and twenty in number, converging to the nipple. These ducts are surrounded by a matrix of fibrous connective tissue, which forms for the breast throughout its career a stable supporting framework. In this condition, illustrated in *Fig.* 62,* it remains through most of the years of childhood. As puberty makes itself felt, the breast is awakened to greater activity. The ducts begin to throw out lateral branches, which in turn subdivide again and again, and in this way a racemose gland is formed possessing an enormous number of small

* All the illustrations in this article have been prepared from drawings made by Mr. J. R. Ford.

terminal acini. Each of the main ducts opening at the nipple forms as it were the stem of a tree, of which the terminal acini may be called the 'leaves', and each mammary tree so constituted occupies a so-called lobule of the breast—so-called, because each lobule is not a separate structure which can be distinguished in the living subject, but is rather a histological conception. Meanwhile, as the branches grow, the connective-tissue matrix undergoes a corresponding increase in bulk, so that the glandular tissue is not as a rule to be found encroaching on the cellular tissues in the neighbourhood. It is this connective tissue which is largely responsible for the increase in size at this period. Most of it persists throughout life, even when the glandular tissue has begun to disappear, and I think it is perhaps this *relative* increase in the

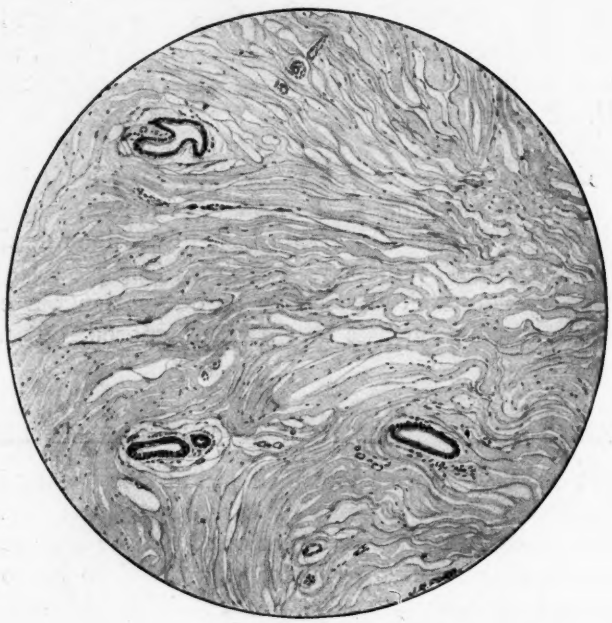


FIG. 62.—Immature breast from a girl, age 11, showing fibrous connective tissue and large ducts only. (× 46.)

fibrous tissue which has sometimes given rise to a mistaken idea that an extensive pathological change, or fibrosis, has taken place. This will be referred to more fully later.

The epithelial lining of the mammary gland is, as has been said, derived from modified sweat glands—ultimately, that is to say, from squamous epithelium—and consists throughout of one layer of epithelial cells, beneath which is a layer of smaller, more or less flattened, supporting cells. In the main ducts the cells of the glandular layer are columnar or cylindrical in shape, and these give way, as the ducts branch, to cells which are more definitely cubical. The transition from columnar to cubical is not abrupt, but very gradual, and it is difficult, or impossible, to say where duct ends and acinus, or true secretory tissue, begins. The truth is, I believe, that all the cells participate in the secretion, and the difference lies rather in the chemical

composition of the secretion than in their morphology. Even in the immature breast, which contains only the large collecting ducts, these ducts are filled with a fluid secretion.

I wish to draw attention to the relation of any group of acini in a normal adult breast to the surrounding connective tissue. It is evident that each group is surrounded by a rarefied form of connective tissue, almost myxomatous in appearance (*Fig.* 63), which is ready, as it were, to receive the enormous growth of gland which takes place before full functioning activity supervenes. This begins to be apparent even in the early months of pregnancy. The terminal acini multiply rapidly. Their lining epithelium swells and almost obliterates the lumen. Fat globules begin to appear in the inner parts of the cells, sometimes even in the cells of the main ducts. At this stage the masses of glandular tissue dominate the picture (*Fig.* 64), and can be seen in sections with the naked eye. When lactation begins, the inner half of the lining cells breaks down and is discharged into the lumen of the acini ; the secretion so formed is forced by the pressure from behind of more secretion into the smaller ducts, and so to the main ducts and their exits in the nipple.

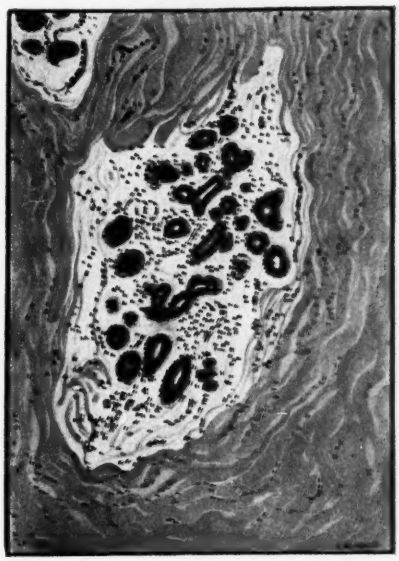


FIG. 63.—Normal group of acini from the breast of a virgin, age 19. (× 60.)

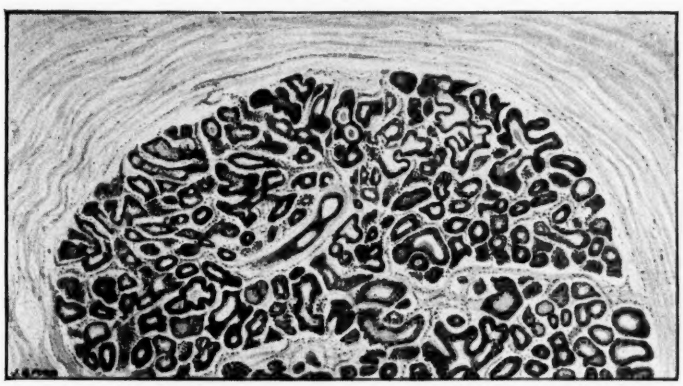


FIG. 64.—Part of a group of acini undergoing normal hypertrophy of pregnancy shortly before the commencement of lactation. (× 60.)

So far nothing has been said of the modifications in shape which are seen in a duct as it passes from the surface of the nipple to the interior of the breast. These changes appear to me to be of great, though imperfectly appreciated, importance in the pathology of the diseases of the breast. I wish to draw

particular attention to the manner in which the ducts open on the surface of the nipple. In the accompanying drawing (*Fig.* 65) it will be seen that the actual mouth of the duct is a funnel-shaped structure lined with ordinary squamous epithelium. These squamous cells extend for a considerable distance below the surface of the nipple, and, in the inactive breast, at the point at which they end—that is, at the neck of the funnel—the lumen of the duct becomes extremely narrow and is usually almost obliterated. Immediately beyond this point there is an abrupt transition from squamous to columnar epithelium, and at the same time the duct suddenly opens out, the lumen becoming wider than at any other part of its course. This part forms the ampulla of anatomical nomenclature. The walls of the ampulla, unless it be greatly distended with secretion, are irregularly folded, and so it can adapt itself to wide variations in content.

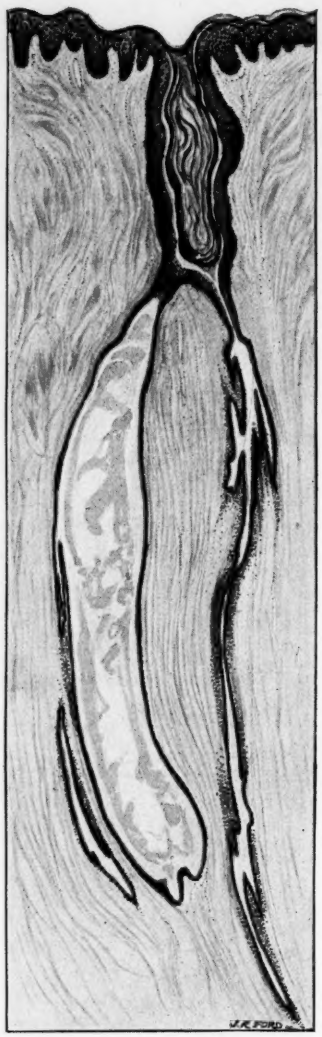


FIG. 65.—Longitudinal section of a duct mouth, into which two collecting ducts open, one being distended with secretion. The epithelial plug is shown in relation to the squamous-celled lining of the mouth. (× 20.)

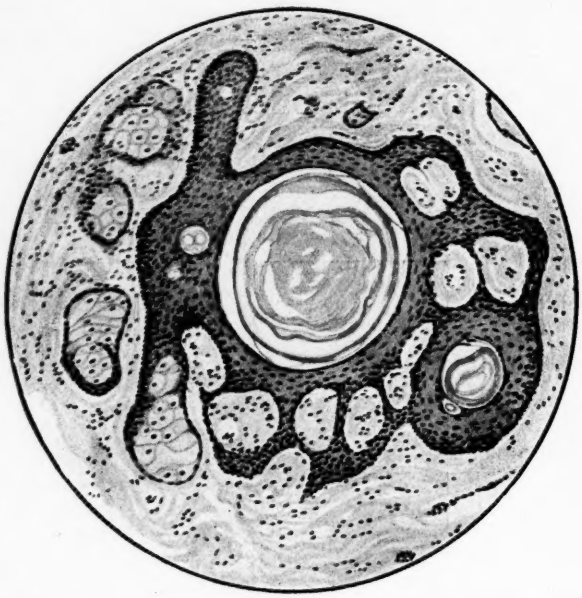


FIG. 66.—Transverse section (paraffin) of the mouth of a duct, showing the laminated epithelial plug in position. (× 100).

A further point which I wish to emphasize is the fact that the funnel-shaped mouth of the duct is lined with an epithelium which normally keratinizes on the surface. These keratinized layers inevitably tend to accumulate in the depression formed by the funnel, and I have found that in the non-lactating breast the mouths of the ducts are, almost without exception,

filled by a laminated plug of keratinized epithelium. I have examined a very large number of nipples, both by making longitudinal sections and by serial transverse sections. In the longitudinal section (*Fig.* 65) the plug can be seen to form a cone-shaped cast of the mouth of the duct. In a transverse section (*Fig.* 66) the laminated plug is well seen, and it must be remembered

FIG. 67.—Near the surface, showing laminated plug and sebaceous secretion.

FIG. 69.—The narrowest part of the duct at the apex of the funnel. The lining cells are of transitional type.

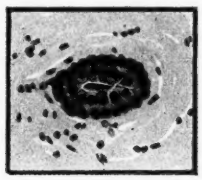


FIG. 70.—Just beyond *Fig.* 69. The lumen still very narrow. The lining cells are definitely columnar.

FIG. 68.—Further from the surface. The lumen is much smaller. The epithelial plug is still present.

FIG. 71.—Immediately beyond *Fig.* 70. The duct has opened out into the ampulla, with wide lumen and irregular outline.

FIGS. 58–62.—TRANSVERSE SECTIONS, SERIAL (FROZEN IN GELATIN), OF THE MOUTH OF A DUCT. (× 160.)

that this is drawn from a paraffin section, in which the plug is somewhat shrunk. There are large sebaceous glands opening into the mouths of ducts close to the surface, and the secretion of these has, of course, been dissolved out. In the above series of drawings (*Figs.* 67 to 71), made from frozen sections stained with hæmatoxylin and Soudan III, both laminated epithelium

and sebaceous secretion are well shown. As soon as the narrow neck of the funnel has been passed, the plug disappears and the duct opens out again, as has already been said.

The mouth of the duct in a lactating breast forms a striking contrast to this (*Fig.* 72). The narrow neck is now widely open, and the epithelial plug, except for a few layers at the periphery, has disappeared. A transverse section taken through the narrowest part shows instead the secretion containing globules of fat lying in a lumen of considerable size (*Fig.* 73).

FIG. 72.—Section of the nipple of a lactating breast, showing the mouth of the duct opened up and the epithelial plug dislodged.

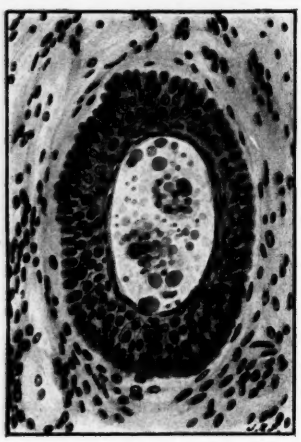


FIG. 73.—Transverse section (frozen in gelatin) through the narrowest part of the duct in a lactating breast. The lumen has opened out and is filled with secretion (*hæmatoxylin and Soudan III*). (× 165.)

Sections of the nipple also show the large amount of unstriped muscle tissue, disposed rather irregularly in small bundles, which this structure contains. The contraction of this muscle is presumably largely responsible for the narrow neck of the funnel. In any case it is clear that no secretion present in the ampullæ of a non-lactating breast is normally allowed to escape. The duct therefore becomes additionally sealed by a firm epithelial plug. This may be accentuated by the retraction or deformity of the nipples which is often found to be associated with chronic mastitis, and is by no means to be regarded as a sign diagnostic of carcinoma.

THE NORMAL PHYSIOLOGY OF THE BREAST.

It may seem at first somewhat superfluous to devote a special section of a paper on the pathology of an organ to a consideration of its normal physiology; but in the case of the breast I believe the latter to be to a large extent the key to its pathology.

I have so far spoken of the breast as a gland either inactive or functioning

—that is, lactating; but I now wish to put forward the view that the breast is a gland which throughout life is exhibiting some secretory activity, the difference between a lactating and a non-lactating breast being one partly of degree and partly of the chemical constitution of the secretion.

The physiological history of the mammary gland begins at birth. It is well known that babies at birth often have a swelling of one or both breasts, and that sometimes a milky fluid may be expressed from the nipple. This may lead in some cases to a pyogenic infection of the breast, possibly with some abscess formation; but there is little doubt that primarily there is an abortive attempt to fulfil its ultimate function of lactation. This may be called clinically an 'infancy mastitis', and is presumably to be explained by the fact that both fœtal and maternal breasts are subjected to the same hormonic stimulus. I have no knowledge of the histological appearance in such a case, but I do know that in many of the sections of breasts of young children that I have seen, the ducts are considerably distended with secretion, which may be taken as evidence that the cells of the immature gland have some functional activity.

The clinical condition of mastitis is not, however, often seen in children between infancy and the age of 11 or 12, but from this time onwards the physiology of the breast is very intimately connected with that of the sexual glands. After the age of 11 in girls and 13 in boys the condition of 'puberty mastitis' is very commonly seen. There is a painful swelling of one or both breasts, which, though not acute, may persist in boys for two or three years and then disappear. In girls it passes into the phase of normal hypertrophy which attends the attainment of sexual maturity. Even in boys, one or both breasts may undergo a similar hypertrophy at a considerably earlier age, producing the condition known as 'gynecomastia'. Occasionally such breasts have been removed, and histologically the appearance of the organ has been found to agree closely with that seen in the immature female breast, the ducts being well developed and distended with a clear secretion; though in two cases recently recorded[1] sections showed also that several of the changes characteristic of chronic mastitis were present.

In girls, with the arrival of sexual maturity the mammary glands enter upon a phase of permanent semi-activity, and are subjected to a constantly recurring physiological stimulation which is repeated in varying degrees at frequent intervals for a period of at least thirty-six years. At every menstrual period the glands are acted upon by hormones from the sexual glands, which awaken some sort of activity in the epithelial cells. In many women this may pass unnoticed. Many others are painfully conscious of the process, and may be led to consult a doctor about it, who will perhaps make a diagnosis of 'chronic mastitis'. The condition should perhaps rather be called at first 'menstrual mastitis', though as time goes on the physiological state becomes more and more difficult to distinguish from the pathological. Apart from this, it is possible that prolonged sexual excitement may stimulate the breast to such a degree that an actual secretion of milky fluid from the nipple is obtained.[2] This is the extreme example of the action of ovarian hormones.

In the event of pregnancy becoming established, the menstrual cycle is for the time being abolished, and a different form of hormonic stimulation is

substituted. In response to this the cells enter upon their full functional activity, and a period of enormous hypertrophy, culminating in lactation, follows. Lactation is succeeded by a process of shrinkage, during which the cells of the hyperplastic gland largely disappear and are absorbed—unless further pregnancies follow in quick succession, in which case the gland may be almost continuously active for several years.

At about the age of 48 the possibility of further pregnancies comes to an end, but the phase of the climacteric may subject the mammary glands to a series of irregular hormonic stimuli, which only cease with the final establishment of the menopause. Then, and then only, are these glands allowed to rest, and I believe that in strict normality an almost complete atrophy of glandular tissue gradually takes place. First the acini disappear, then the small ducts, and finally only the largest collecting ducts remain. Even in these the cells are small, and the lumen greatly shrunken (*Fig.* 74). The connective-tissue framework may also become reduced in bulk, though the external appearance of the breast would often belie this owing to its becoming infiltrated with fat.

FIG. 74.—Normal atrophy after the menopause. The acini are very shrunken and their lumen is almost obliterated. (× 60.)

After the menopause, therefore, and in old age, there is no true physiology of the breast to record, but too often a physiological process has become merged in a pathological condition, which probably has had its origin many years before. A physiological stimulus may amount to a pathological insult if only it is repeated often enough.

It may be asked why I have chosen to dwell in such detail upon a series of physiological commonplaces. My object has been to emphasize the fact that the epithelial cells of the breast are producing some sort of secretion almost continuously for many years. I have already demonstrated that, except during lactation, the mouths of the ducts are not normally patent, and that no secretion can escape. The physiology of the breast entails, therefore, not merely secretory, but also continuous absorptive, activity. I shall return later to the bearing of this upon the pathology of chronic mastitis.

THE HISTOLOGICAL EVIDENCE OF MASTITIS.

I now turn to a description of the histological changes which I believe to be indicative of the presence of chronic mastitis. I can but describe each one in turn; it must be understood, however, that all may be seen at once in one small area, though as a rule their relative distribution in the breast is very erratic.

If, as the name of the disease implies, it is an inflammatory condition, then it would be expected that one of the cardinal signs of inflammation,

infiltration of the connective tissues with leucocytes, would invariably be present. Actually, the presence of round cells is not an invariable sign, or, at any rate, all parts of the diseased breast are not equally affected. In the earliest stages the infiltration is most commonly seen in the loose connective tissue immediately surrounding the groups of acini, but the cells are usually not at all densely packed. The presence of a few appears, indeed, to be normal in the adult breast (*Fig.* 63), and their number is always much increased in the lactating gland. In the later stages of mastitis the round cells tend to be concentrated at particular points, in close relation to dilated acini or along the course of ducts; but often it is not at all clear why groups of cells are present at one point and entirely absent in other places that are, to the eye, equally, or even more, affected by the disease.

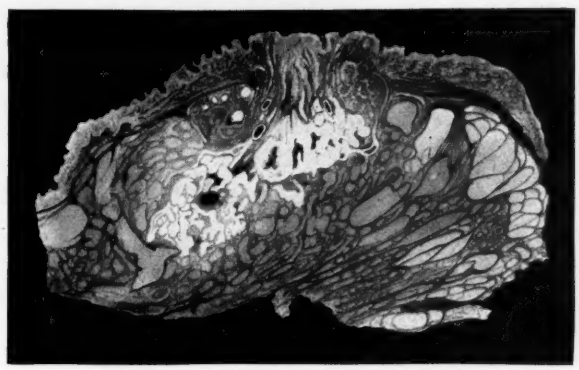


FIG. 75.—Acute stagnation mastitis in a breast already the seat of chronic mastitis. Inspissated milk can be seen apparently infiltrating the tissues in the centre, and all the ducts and acini are distended with secretion. The nipple is retracted.

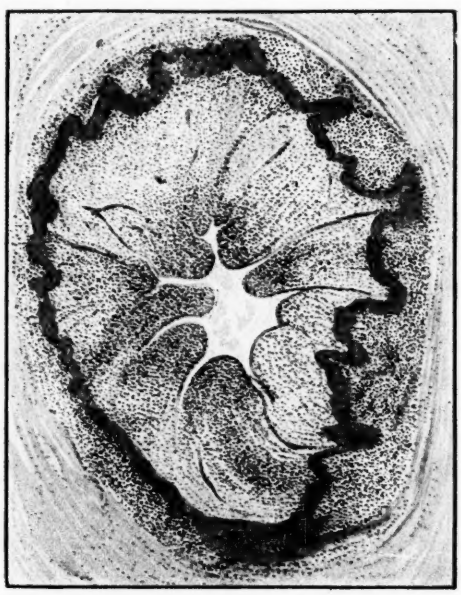


FIG. 76.—Acute stagnation mastitis, from the same specimen as *Fig.* 75. Round-celled infiltration, fibrosis, and destruction of the epithelial lining. (× 25).

It is, however, evident that the round-celled reaction is more intense when the lumen of the acini or ducts that they surround is filled with a fluid rich in disintegrating cells or fat-containing secretion. The mere presence of epithelial changes certainly does not on the whole tend to determine a concentration of leucocytes. It is the presence of dead cells or inspissated secretion which attracts them most strongly. The microscopic appearances suggesting *inflammation* are therefore indirectly produced by interference with the escape of the products of epithelial activity from ducts and acini. The most extreme condition of leucocytic infiltration that I have found was in the breast of a woman who became pregnant after having developed an advanced chronic mastitis. The breast (*Fig.* 75) attempted to lactate, but the secretion was unable to escape normally, and reabsorption of the milk

was only effected with an intense inflammatory reaction, which resulted in the destruction of the epithelial lining of many of the ducts, with some surrounding fibrosis. The peculiar appearance produced is shown in *Fig.* 76. A less acute effect is seen in *Fig.* 77. In this case the patient, who was suffering from chronic mastitis, became pregnant, but normal lactation hypertrophy did not take place, the result being the appearance shown in the section. A similar condition, which may be called a 'stagnation mastitis', is seen clinically in a woman who has for some reason been compelled to cease suckling her child soon after its birth. Reabsorption of secretion is then attended by acute pain and inflammation, which may initiate a chronic mastitis and give rise to considerable suffering in a subsequent pregnancy.

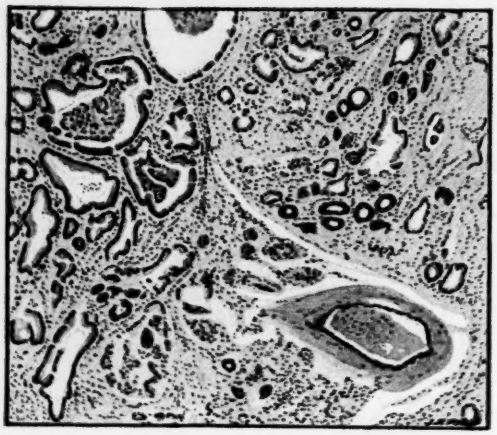


FIG. 77.—Attempted lactation in the presence of an advanced chronic mastitis. The acini have not developed normally. There is round-celled infiltration, fibrosis, and some epithelial proliferation. From a patient, age 43, who died immediately after the birth of her first child. (× 60.)

It is important to note what type of cell is most commonly found, and it is clear that the lymphocyte nearly always predominates. In the extreme case mentioned there were also a good many polymorphonuclear cells, and in another (*Fig.* 95) there were large numbers of eosinophil cells, producing a very unusual appearance (*Fig.* 78).

FIG. 78.—Section from the breast shown in *Fig.* 95. It is infiltrated with eosinophil cells. (× 300.)

It is also possible by suitable staining to demonstrate in most cases a sprinkling of the so-called plasma cells outside the ducts and among the acini. These tend to be more numerous when the leucocytic infiltration is most marked, but it would be wrong to attach too much significance to their presence. They are probably the normal precursors of fibroblasts, and are concerned only in the production of the fibrosis next to be described.

The presence of fibrosis is another change seen in the interstitial tissues which is very capricious in its distribution. The word has, I suspect, been

used very loosely by some writers on the subject, and sometimes the normal fibrous stroma has been mistaken for a pathological fibrosis owing to the alterations in the relative amounts of fibrous and glandular tissue. In the breasts of some women who have passed the menopause, glandular tissue has largely disappeared and only the larger ducts with a few groups of shrunken acini remain. These appear as oases in a desert of dense connective tissue, but there has not necessarily been any fibrosis in the sense of replacement of glandular by fibrous tissue, or even any overgrowth thereof (*Fig.* 74). When this does occur the histological appearance is characteristic.

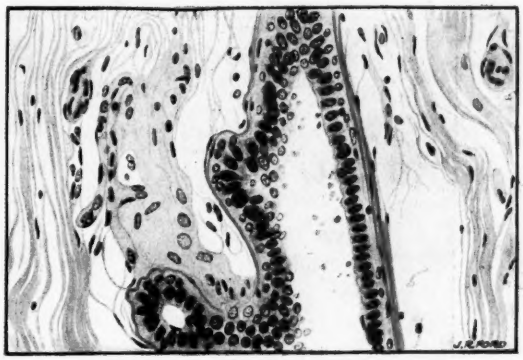


FIG. 79.—Fibrosis, early stage. Increase of fibrous connective tissue immediately outside the acini and ducts. (× 210.)

Often the first evidence of true fibrosis is seen as an increase in the density of the layer of fibrous cells immediately outside the ducts or acini. In sections stained with eosin this is seen as a conspicuous red rim round these structures, consisting of several layers of connective-tissue cells. It is well shown in *Fig.* 79.

In another form of fibrosis the area of loose and transparent connective tissue round the groups of acini tends to become contracted or to disappear altogether. The eosin staining then extends evenly right up to the acini (*Fig.* 80). In a later stage the epithelial cells may disappear and be replaced entirely by fibrous tissue arranged as a group of small whorls, each whorl presumably representing an acinus. This appearance tends to resemble the arrangement of fibrous tissue seen in a fibro-adenoma intracanaliculare, and that, after all, is probably the late stage of an originally glandular tumour in which the fibrous tissue has become predominant at the expense of the epithelial cells.

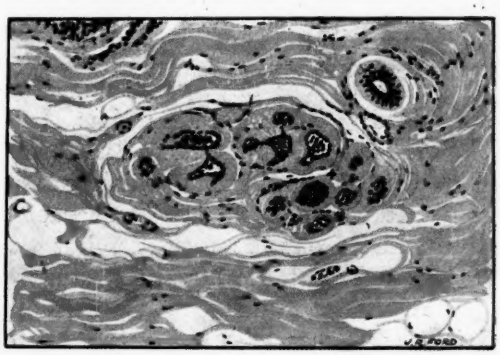


FIG. 80.—Fibrosis, later stage. Replacement of loose connective tissue with denser tissue. (× 100.)

Occasionally it may happen that there is an actual hypertrophy of fibrous tissue throughout the breast, and I have a specimen which consists chiefly of a large fibrous mass, almost resembling a fibro-adenoma of unusual size (*Fig.* 81). The other breast was the seat of a very advanced type of chronic mastitis with cysts, and both were removed at operation. This massive fibrosis is, however, an unusual condition. Finney, indeed, says of it that it is " certainly extremely rare."[3]

Dilatation of ducts and acini is a change that almost always accompanies chronic mastitis, but it is difficult to judge at what point the process is to be regarded as pathological. For it is quite normal to find a dilatation of the large collecting ducts and ampullæ which can be seen with the naked eye when

FIG. 81.—Massive fibrosis of the breast.

the breast is sliced, and this is only to be expected in a secreting gland which has no outlet except when lactating (*Fig.* 82). As the secretion accumulates, this distention may be communicated to the smaller ducts and finally to the acini, which normally have only a very small lumen ; but clearly there is no criterion indicating definitely the point at which the process becomes pathological. I have therefore not been able to use any particular standard. Each section has been judged on its merits. The formation of small cysts which can be felt with the fingers is characteristic of chronic mastitis, but this is only a more advanced stage of a change which can at first only be appreciated under the microscope. The presence of large cysts lined by flattened epithelium and filled with a clear greenish fluid, a turbid milky fluid, or a cheesy mass, is also characteristic of a still later stage of mastitis. These advanced changes are presumably associated with obstruction to a duct by fibrosis, cellular proliferation, or pressure ; but I have not been able to demonstrate this in relation to any particular cyst.

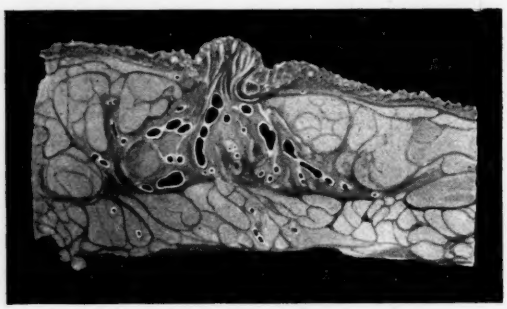


FIG. 82.—Normal breast somewhat atrophied, showing dilated collecting ducts.

The epithelial changes in chronic mastitis are more definite and more easily identified than those described so far. The changes appear to me to be of two different types. In one the change is primarily an enlargement

or swelling of the cells without very much active proliferation; in the other the cells do not always undergo any alteration in size, but simply proliferate in varying degrees, forming either the 'laciform proliferation', so named by Sir Lenthal Cheatle, or 'massive proliferation', solid lumps or columns of cells filling and distending the spaces originally occupied by the acini or ducts in which the process arises. The first change may be partly degenerative, but it is not connected with the so-called 'involution changes' seen in the breasts of women who have passed the menopause, for I have found it at all ages from 16 upwards. Nor do the cells show any fatty changes in their cytoplasm when tested with the special fat stains. It seems, on the other hand, to be associated with some form of irritation, and may occur in patches. This is well seen in *Fig.* 83, where an early stage is illustrated in a longitudinal section of a duct. In certain places the normal low columnar cells have enlarged and become more than double their usual height. The lumen of the duct is at the same time somewhat distended, and outside the duct in relation to the altered cells is some degree of infiltration with round cells. Further stages of the same change are seen in *Figs.* 84 and 85. In these sections, represented on a small and a large scale, the change is clearly associated with ducts or acini which were already distended, some showing the presence of secretion (*Fig.* 85). This is nearly always found to be the case. In one duct (*Fig.* 84) the cells are tending to disintegrate, and it is possible that these changes may in some cases precede

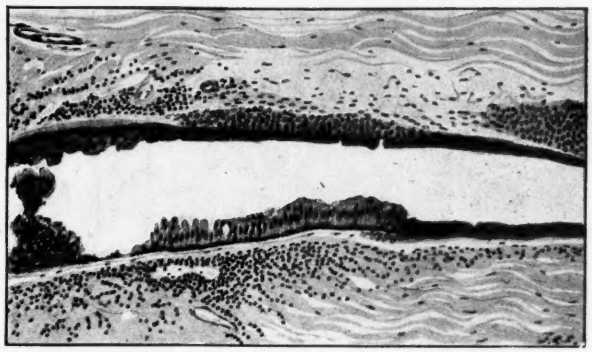


FIG. 83.—Longitudinal section of a duct. Swelling of epithelial cells in patches, with round-celled infiltration outside. (× 100.)

FIG. 84.—Swelling of epithelial cells in some of a group of acini and ducts; distention of others. (× 100.)

the formation of a smooth-walled cyst, lined with flattened cells representing the basal layer only, and filled with milky fluid containing much cell débris; but I have not been able definitely to connect these changes with the other.

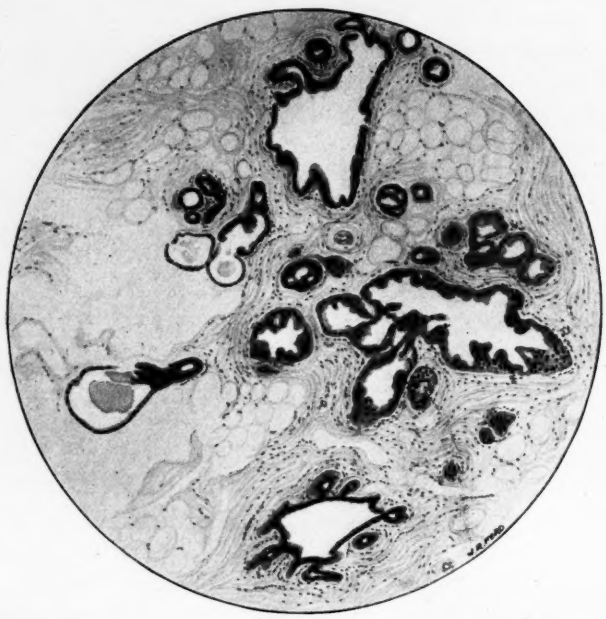


FIG. 85.—Distention of a group of ducts and acini, with swelling of the epithelium in some. (× 36.)

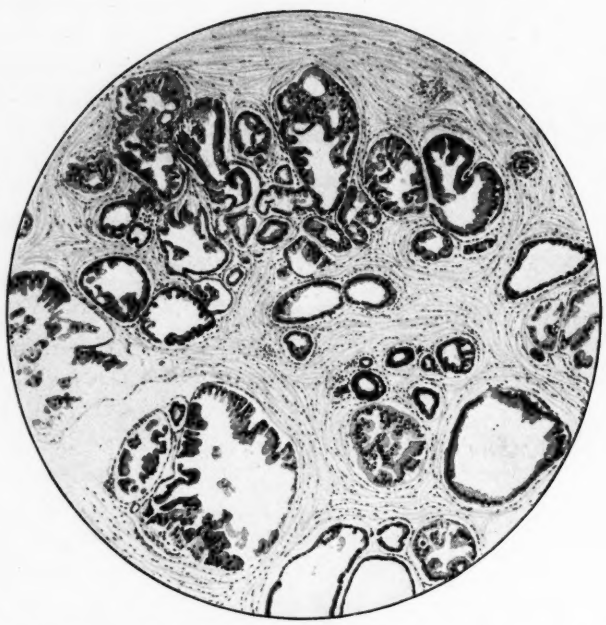


FIG. 86.—A further degree of the same change as is seen in *Fig.* 85. Apparently some proliferation. (× 46.)

In most instances the change seems to spread and involve the glandular tissue of neighbouring groups, until the appearance seen in *Fig.* 86 is produced, or it may become even more striking than this. It is to be noticed in these three figures that there is no sign of a round-celled infiltration; but, as has already been pointed out, this change is very capricious, and its absence can occasion no surprise. The appearance of proliferation is in these sections probably due in most cases to oblique cutting of the irregularly swollen cells, but there does occasionally seem to be a true proliferation associated with it. The appearance in these sections is to be contrasted with that in *Fig.* 87, which probably represents a true senile change, and was found in the breast of a woman of 70. The cells are here coalescing, and the nuclei have become very irregular in size.

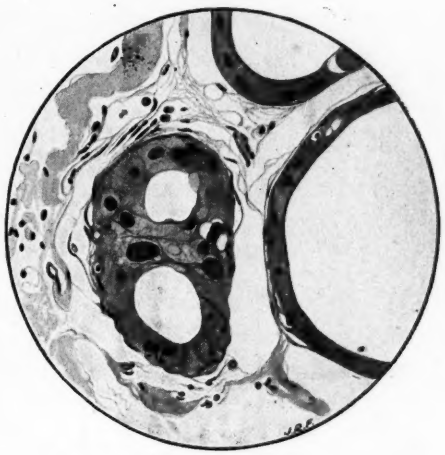


FIG. 87.—Section from a senile breast, most of which showed atrophy only. The cells are enlarged and are coalescing. The nuclei are very irregular in size and position.

The second type of cellular change is, as I have said, primarily a proliferative change, and clearly indicates a much greater activity in the cells. An early stage of this is represented in *Fig.* 88. Most of the ducts and acini

FIG. 88.—Irregular proliferation of epithelial cells, with patches of round-celled infiltration. Some normal acini remain. (× 74.)

in neighbouring groups have been obliterated by the proliferation of their cells, and in this instance the change is accompanied by a well-marked degree of round-celled infiltration in the neighbourhood. A single duct is seen on a

larger scale in *Fig.* 89. Here, as proliferation proceeds, some of the cells are being cast off and will disintegrate. When these have disappeared the laciform appearance will presumably result. This is not in any essential particular different from the massive proliferation seen in *Fig.* 90, where the outlines of a few acini can still be made out.

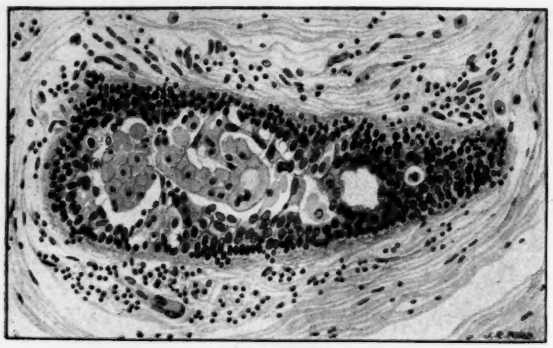


FIG. 89.—Proliferation of cells in a single duct, with desquamation of some cells. (× 133.)

Another change which seems to be closely associated with the cellular changes already described takes place chiefly in the larger ducts, and results in the formation of true papillomata. The first stage of this is shown in *Fig.* 91, where the wall of a distended duct is seen to be sprouting small excrescences all round its circumference. A further stage is shown in *Fig.* 92. The excrescences are becoming more complex, and in the upper part a large papillomatous growth is in process of formation. The final stage is shown in *Fig.* 93. A large papilloma lies in a smooth-walled cyst attached by a stalk at one point, which is not seen in the section. Such papillomata are commonly found in the ducts close to the nipple, and their presence often gives rise to a clear or blood-stained discharge from the nipple, and this usually attracts the patient's attention. But I do not believe that such a papilloma ever occurs as an isolated phenomenon. The signs of chronic mastitis would certainly be found in other parts of the breast if search were made for them.

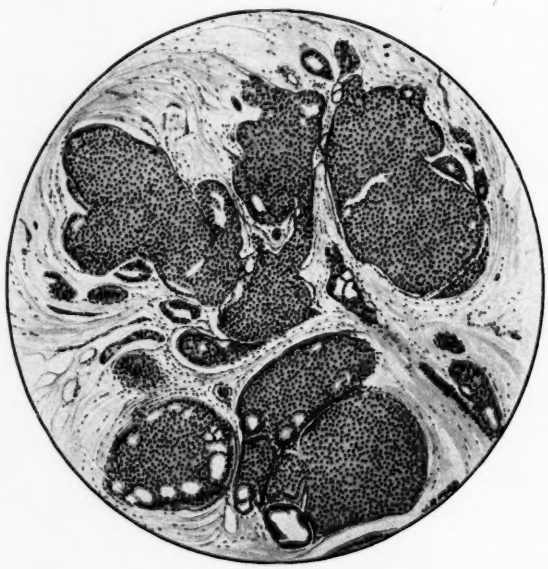


FIG. 90.—Massive epithelial proliferation in an advanced stage. A few acini can still be seen. (× 80.)

I have now given some account of all the histological changes seen in chronic mastitis. Summarized in as few words as possible, they are: round-celled infiltration, fibrosis, dilatation, epithelial changes of two kinds, and formation of papillomata. In the early stages of the disease some degree of fibrosis, as described here, some degree of dilatation of the small ducts or acini, and a few patches of round cells

are the chief histological evidences of its presence. In the later stages all these changes become accentuated and are associated with one or both of the forms of epithelial change. Sometimes the leucocytic activity appears not to be accentuated at this stage, but may, on the other hand, be almost absent. Numerous

FIG. 91.—First stage of the formation of papillomata in a duct. The epithelial cells are swollen. (× 74.)

FIG. 92.—Further stage in the formation of a papilloma. (× 44.)

'window-pane sections' would probably reveal their presence in certain places, but of this I cannot be sure. In the stage at which operation is performed, the slight dilatation has usually culminated in the formation of large cysts, but at this point the condition is given a number of different names indicating supposedly distinct diseases. This will be referred to again. The laciform or massive proliferation abundantly indicates that a profoundly abnormal activity has by some means been induced in the epithelial cells, and this may be of the greatest importance in considering the question of malignancy. This also will be discussed later.

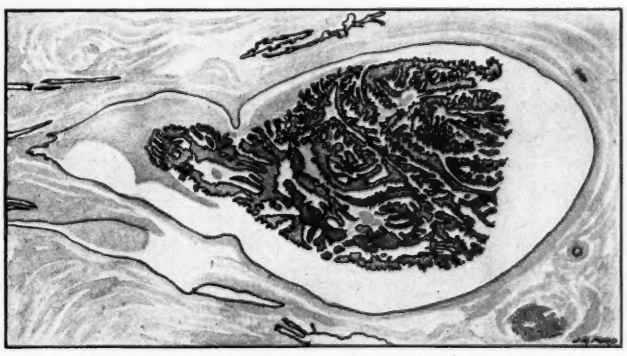


FIG 93.—Fully formed papilloma lying in a smooth-walled cyst. The stalk is not seen in this section. (× 8.)

THE AGE AND SEX INCIDENCE OF PATHOLOGICAL CHANGES IN THE BREAST.

One object of this investigation was to ascertain if possible at about what age the changes of chronic mastitis first became apparent. It is difficult, however, to obtain accurate facts from statistics based on the relatively small numbers to which I was necessarily limited. The post-mortem room material provided 116 specimens, of which 57 were normal and 59 showed inflammatory lesions. It would clearly be quite misleading to express the incidence of chronic mastitis as a percentage of the whole, since older patients would inevitably preponderate in material gathered from this source. I have therefore attempted to eliminate this, though only partially, by taking the age of each case and showing in the accompanying diagram (*Fig.* 94) the percentage,

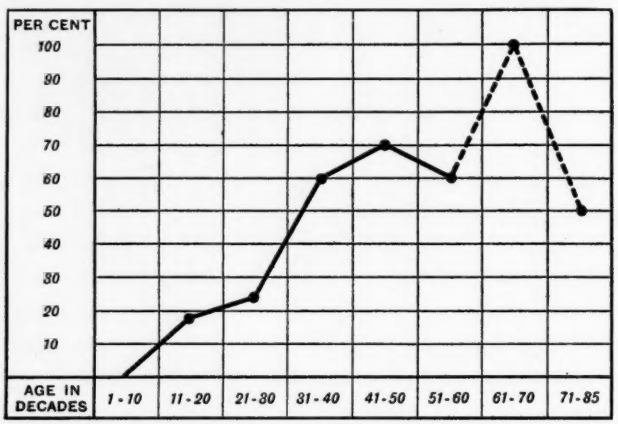


FIG. 94.—Chart showing age incidence of chronic mastitis.

in every decade, of individuals showing positive signs of chronic mastitis. It will be seen that the lesions first become apparent in the second decade, and rise to a maximum in women aged 40 to 50. It is, of course, possible that the figure of 18 per cent in the second decade would be lower if I had more material on which to base it. Also I feel quite sure that the extreme irregularities seen in the graph after the age of 60 are quite misleading, the number of cases beyond this age that were investigated being very small. I have therefore shown this part only in dotted lines. It is evident from this diagram that chronic mastitis is not a disease of the climacteric only, as has sometimes been supposed. It may have had its beginnings thirty years before this, though it is most apt to become clinically obvious at the later age.

The post-mortem room material also gives some indication of the influence of lactation upon the incidence of chronic mastitis. Details of family history had not been recorded in all cases, but the figures as far as they could be ascertained were as follows :—

POST-MORTEM ROOM MATERIAL.

Average age of all positive cases,			unmarried or childless	43 years.
,,	,,	,,	parous women ..	51 ,,
,,	,,	,,	.. .. ..	47 ,,

A similar impression is obtained from the series of cases which have developed manifestations of mastitis necessitating operation. The youngest unmarried patient in this series was 24. The youngest married patient was 46. The average ages were as follows :—

OPERATION MATERIAL.

Average age of	unmarried patients	37 years.
„ „	married patients	46 „
„ „	all patients	43 „

It seems to be clear that chronic mastitis tends to appear some *ten years* earlier in single women than in those who have borne children.

I have so far referred only to the incidence of chronic mastitis in the female sex. It is well known, however, that males may suffer from all the same affections of the mammary glands as females, though very much less commonly. The early physiological types of inflammation in the breast, namely infancy and puberty mastitis, occur almost as often in boys as in girls. Afterwards the breast in the male becomes quiescent. I need not here consider the condition known as 'gynecomastia', in which a boy, even before the period of puberty, develops a mammary appendage of the female type. In some cases this is more apparent than real, the hypertrophy being due chiefly to a local excess of fat. In others there is an actual development of immature glandular tissue resembling that of a girl. This, although abnormal, may be purely physiological and have no connection with mastitis. In later life, however, men may develop in their breasts all the same conditions as are seen in women. Ordinary chronic mastitis is not very rare in old men. Sometimes cysts, papillomata, and so forth are developed, and may necessitate operation. A definite percentage of cases of carcinoma of the breast is found in men. Many studies of the diseased conditions found in the male breast have been published,[4] but these do not reveal any condition different from those found in women. I thought it worth while, therefore, to examine a series of male breasts from the post-mortem room, in order to find out how often the changes of chronic mastitis were to be found. I have examined 37 specimens, and found some changes in 4 of them. In two cases there was localized epithelial proliferation only. In one there was fibrosis taking place in the wall of a dilated duct. In one from a man of 38 the picture was that of a well-developed chronic mastitis. Some of the ducts were enormously dilated, and the secretion contained large numbers of cells. In some places there was swelling and desquamation of the epithelial cells, and there were numerous patches of round-celled infiltration. The average age of the four patients was 54.

It was noticeable that almost every specimen showed that the ducts had a wide lumen and contained secretion, so that the conditions very closely resemble those found in women. It must be remembered, however, that the gland tissue in the male is represented only by the largest collecting ducts. In the female breast the changes of mastitis are seldom seen in these large ducts, but are almost always in the smaller ducts or acini, which do not exist in the male. It may be, therefore, that the epithelium of the large ducts is for some reason less vulnerable, and so less liable to pathological changes,

than that of the more truly glandular tissue. Perhaps it is partly to this that the male breast owes its comparative immunity from disease. It is also less subject to irregular hormonic stimuli.

THE THEORIES OF CAUSATION.

As indicated at the beginning of this paper, the idea of the causation of chronic mastitis that has commonly held the field is one involving the presence of some obscure bacterial infection. Sir Lenthal Cheatle[5] has indeed gone so far as to suggest that epithelial changes, and even carcinoma itself, may be initiated by irritating foreign substances which have found their way into the breast through the openings in the nipple. Foreign substances so introduced can scarcely be supposed to be sterile, so that the conception again brings in the idea of direct bacterial infection from the outside. I cannot, however, discover any evidence in favour of the view that the inflammation is bacterial. All that I can gather seems to show that bacteria take no share in the disease.

I have shown, I believe conclusively, that the normal breast has, except during lactation, no outlet through the nipple. The mouths of the ducts are mechanically closed a little way below the surface by muscular contraction which reduces the lumen almost to nothing. They are in addition sealed by a firm plug of epithelium, reinforced by sebaceous secretion, a substance which is known to be unfavourable to the growth of bacteria. It seems very unlikely, therefore, that any foreign substance could obtain entrance through the nipple. It cannot be claimed that lactation affords an opportunity for infection from without, since the changes of chronic mastitis are so often found in breasts which have never lactated.

There is the alternative supposition that bacteria may have been brought to the breast by the blood-stream. In the case of a tuberculous infection this undoubtedly happens, and presumably a pyogenic infection may occasionally occur in the breast in the course of a blood infection, as may happen in any other part of the body; but it is difficult to believe that every one of the vast number of women afflicted with chronic mastitis has pyogenic bacteria circulating in her blood in numbers large enough to produce a diffuse lesion in both breasts.

In either case it should be possible to recover these bacteria from a breast showing a well-marked degree of mastitis; but this I have failed to do. On many occasions I have made blood-broth cultures of breasts showing the changes of chronic mastitis, pieces of tissue having been taken from the breast with all aseptic precautions immediately after its removal from the body at operation. These have been incubated both aerobically and anaerobically. I have also made cultures of the fluid contents of cysts and of the milky fluid which can often be expressed from the dilated ducts of a breast showing chronic mastitis, a fluid which, obtained from cysts large or small, is apt to be erroneously recorded as 'pus' without being further investigated. In no case has any growth of bacteria taken place—a result which an expert bacteriologist might be inclined to attribute to a failure on my part to find a suitable culture medium. This may be so; but further investigation must be left to the expert bacteriologist himself. These negative results are supported by the fact that I have never succeeded in demonstrating the presence of bacteria

by Gram's stain in sections of breast tissue or in the fluid contents of cysts. I may further point out that the histological appearances do not suggest the presence of bacteria. Bacterial infection is almost invariably accompanied by a leucocytic reaction. This, as has been said, is often absent in chronic mastitis. Also the leucocyte characteristic of pyogenic infection is the polymorphonuclear cell. In chronic mastitis it is always the lymphocyte which is present, almost to the exclusion of polymorphonuclear leucocytes. Nothing can be inferred from the presence in one case of large numbers of eosinophil leucocytes.

I have mentioned that enlargement of the axillary lymph glands is often associated with chronic mastitis, but this cannot be interpreted as necessarily indicating bacterial infection. Absorption of any toxic products, bacterial or non-bacterial, may produce signs of lymphadenitis in the glands draining the affected area.

It seems reasonable, therefore, to reject the bacterial theory of causation on the ground that there is no evidence to support it.

Another theory which has been advanced suggests that chronic mastitis may be caused by a general toxæmia, the source of the toxin being a site of bacterial activity in some part of the body. Thus, it was observed by C. B. Lockwood[6] many years ago that the symptoms of an inflammatory tumour in the breast disappeared after the removal of a source of infection, such as an infective vaginitis. But this result would have to be obtained in a considerable number of cases, and full details as to the accompanying circumstances, including the menstrual history, would have to be recorded before this could be regarded as more than a coincidence.

More recently, affections of the mammary gland have been connected with intestinal toxæmia by W. S. Bainbridge.[7] This author recorded in 1921 a series of 25 patients in whom a 'lumpy condition' in one or both breasts appeared to be benefited by treatment of a co-existing intestinal stasis. It is possible that a condition of chronic intestinal toxæmia might influence both the secretory activity of the mammary epithelium and the physiological condition of the patient. From some of these patients, however, cysts or fibro-adenomata were removed by operation, and it is not clear whether improvement in the 'lumpy conditions' of their breasts, which took place after short-circuiting operations upon the large intestine, is to be regarded as an event *post hoc* or *propter hoc*. Judgement as to the possible connection between chronic mastitis and chronic toxæmia must be suspended until more precise evidence is forthcoming.

Another condition which has been held by many writers to be responsible for chronic mastitis is one that is termed 'involution changes'; but this is a phrase which in this connection does not convey any very clear meaning to my mind.

Involution is a word which presumably implies a folding up of something, and as applied to a breast is most properly used when the hypertrophied organ begins to decrease in size and activity at the end of lactation. This change is probably a passive one in most respects, and according to the most recent view[8] results partly from a lessened blood-supply, and partly from the pressure produced by the secretion which is no longer removed as it is formed.

This effect may be aided by bandaging. Under these conditions the epithelial cells undergo autolysis or self-digestion, the products of this metabolism being then removed by way of the lymph- or blood-stream. In this way the hypertrophied organ is removed piecemeal in a relatively short time, until nothing but the ducts with their normal proportion of branches and acini remain. It is difficult to see how any abnormal cellular processes could be initiated in this manner.

It is often stated also that involution changes take place at the menopause, and this statement is associated with the quite erroneous dictum that chronic mastitis is ' a disease of the climacteric'. It is true that the symptoms of the disease are clinically most obvious at that time, because the cause of the mastitis, whatever it may be, has by then been in operation for a number of years, and also because the breast is at that time being subjected to very irregular hormonic stimuli. The disease must in all cases have begun many years before this, and I see no justification whatever for supposing that the 'involution changes' attending the menopause have any connection with the disease. Moreover, these so-called involution changes are, as I have so often seen them under the microscope, usually nothing more than a simple atrophy. After the menopause the epithelial cells are no longer subjected to any hormonic stimulus, the blood-supply tends to become smaller, and the acini in consequence undergo a progressive shrinkage (*Fig.* 74) In a breast which has not developed any marked degree of chronic mastitis with cyst formation or abnormal epithelial activity, the acini become smaller and smaller, the lesser ducts tend almost to lose their lumen, and finally most of the glandular lobules disappear altogether. The large ducts always remain, and usually contain secretion ; but, except for these, a normal senile breast consists almost exclusively of fibrous connective tissue, which in stout subjects becomes infiltrated with large quantities of fat. Abnormal epithelial changes may occasionally be seen in a senile breast which is in most respects normal, and an example of this has been already illustrated (*Fig.* 87) ; but my interpretation of the appearance is only tentative.

There does not seem, therefore, to be much evidence in favour of the involution theory of chronic mastitis, and I think it should be abandoned.

Finally, it has been suggested that trauma may have some influence in initiating a chronic mastitis. I have shown, however, that the changes seen in this disease are diffuse, and clinical experience teaches that the condition is very often bilateral. It is further difficult to suppose that any woman could pass forty years of her existence without being subjected to the ordinary accidents of everyday life, so that a history of trauma can always be obtained by questioning closely enough. Trauma is, in fact, so vague a factor that I do not think it need be further considered.

THE CAUSE OF MASTITIS TO BE FOUND IN PHYSIOLOGICAL PROCESSES IN THE BREAST.

My consideration of the possible causes of chronic mastitis has been so far purely destructive. I now wish to offer a constructive solution of the problem, which is, however, not yet susceptible of proof. Causes outside the breast do not seem to offer any real help. May there be then any cause within

the breast itself? I think the answer to this question is that there may indeed be a cause within the breast arising in the first place from an inherent defect in its anatomical construction, which reacts in its turn upon the normal physiological mechanisms.

It has already been demonstrated that the non-lactating breast is an organ which is subjected to continual physiological stimuli, but which has no outlet for the products of its own activity. A reabsorption of secretion must therefore continually be taking place. In addition to this the epithelial lining of both ducts and acini is being constantly renewed and effete cells are being cast off into their lumina. These cells disintegrate and must somehow be removed unless the breast is to become a stagnating mass of epithelial débris.

All the available evidence seems to me to point to the fact that the breast is itself continually pouring into the lumen of its ducts and acini a possible source of irritation to which all the pathological changes that I have reviewed may be attributed. If there should be a partial failure in the process of reabsorption of fluid and scavenging of epithelial débris, then any such irritation will become accentuated. This in its turn will further interfere with absorption and may initiate abnormal cellular activity, and so a vicious circle will become established.

I do not pretend to be able to suggest exactly what factors in the body may disturb the physiological balance of secretion and reabsorption. In some individuals the balance may be maintained throughout life. In others —probably the majority—it is upset after being successfully maintained for a considerable number of years; even then there are very wide variations in the degree of irritation produced, and this variation occurs not only as between individuals, but also in different parts of the same breast. The variation may be both in the concentration of the irritants formed in the secretions and in the susceptibility of the cells exposed to its action. The reaction of one individual may lie chiefly in the direction of fibrosis; in another it may produce the various forms of abnormal epithelial activity which have already been illustrated. In one case the results of irritation may become apparent after it has been acting for a relatively short time; in another it may be thirty or forty years before any pathological change can be appreciated. But whatever the source of irritation may be, it is clear that the time factor is of great importance. It has been very commonly observed that an advanced degree of chronic mastitis is seen at an earlier age in women who have never borne children than in women who have passed through the healthful process of pregnancy and lactation, and this is confirmed by the figures already recorded. The explanation of this is to be found in the fact that during lactation the mouths of the mammary glands become unsealed, and the products of activity are removed. If a woman has a large family of children, this natural drainage is established with intermissions for a period of many years, and only as middle age approaches do the breasts again begin to feed upon themselves. For this reason chronic mastitis has been called a disease of the climacteric; but the condition really had its first beginnings when lactation ceased for the last time, perhaps ten years before.

It should be possible to test the validity of this reasoning by examining the conditions found in the mammary glands of some other animal, such as

the cow, which is normally lactating throughout its life after sexual maturity has been attained. I have been unable to find in text-books of veterinary science any reference to mastitis in a cow other than the acute infective form, and this is what the hypothesis requires. It is difficult to test the converse of this, that is, to investigate the conditions present in the inactive state, for the mammary gland of an elderly unmarried cow is not easily to be found. The determination of the incidence of chronic mastitis in cows may in fact be regarded as a hopeless quest.

It is worth remarking that recent investigation of the functions of the lymphocyte, the cell so characteristic of chronic mastitis, tends to confirm indirectly this interpretation of the source of irritation in the breast. The lymphocytes in chronic mastitis are, as has been said, very erratic in their distribution, but they are in maximum concentration in a lactating breast when there is any interference with the free escape of milk, and apart from lactation tend to be most numerous in relation to ducts or acini containing much cell débris or fatty secretion. This gains significance from the conclusion arrived at by Dr. S. Bergel[9] that lymphocytes contain a lipase and are concerned in the digestion of fat and lecithin bodies both in physiological and pathological conditions. The presence and distribution of lymphocytes in the breast thus finds a natural explanation.

This hypothesis was first suggested to me by the observation that the non-lactating breast, though a secreting gland, normally possesses no outlet. I revolved the idea for a long time in my mind and tested it in its different aspects before making any close investigation of what others had already written on the subject. When I did so I was interested to find that the same suggestion as to the effect of stagnating secretions in the breast had been made by Bertels[10] in 1913 in the course of a discussion of the relation of chronic mastitis to carcinoma. Bertels' suggestion was quoted and amplified by Lukowsky[11] in 1921; but so far as I can discover the problem has not been examined in detail anywhere else. Binnie's[12] view of the problem is cautious but suggestive: "Chronic cystic mastitis: this pathological process impresses me as a reaction to some irritant. Microscopically and in addition to the parenchymatous changes, there is evidence of reaction in the stroma of the breast. No relation between this disease and any micro-organism has yet been established."

Many very suggestive observations are to be found in a valuable, though now almost unknown, work published by Charles Creighton[13] in 1878. Creighton had noticed that "the excessive production of the secretions of the breast, or their production out of season or their retention at or near their place of origin, will be found to be among the chief factors in the causation of tumours of the breast." He had also noticed in sections of the breast of a ewe that was killed three weeks after giving birth to a dead lamb, and that had not been milked, the enormous number of lymphoid cells which were to be found "in the acini, in the spaces immediately outside them, and in the interlobar fibrillar tissue". He further drew attention to the fact that the periodicity of the mammary gland is its earliest characteristic, and that this begins with its existence as a distinct organ. The first statement of the original law of periodicity he attributes to Shakespeare—"And so from hour

to hour we ripe and ripe, and then from hour to hour we rot and rot."[14] His book contains other pregnant remarks too numerous to mention here.

THE CHEMICAL PROBLEM.

This explanation of the cause of chronic mastitis in the human being is based entirely on deductive reasoning; but a little thought will show that the hypothesis fits in with all the observed facts, both clinical and histological. It cannot, however, be finally accepted until the factor actually responsible for producing a state of irritation has been determined. This introduces an exceedingly difficult problem in biochemistry which can only be dealt with by those who have been specially trained in this line of investigation, and so far as I know little has been done as yet in this direction.

It can easily be demonstrated that there are wide variations in the composition of the fluid which is contained in cysts of the breast, or can be expressed from the cut surface of the gland. Commonly a clear green fluid is obtained; sometimes it is brown. Sometimes the fluid is turbid with cell débris; sometimes it is milky and contains both cell débris and fat globules. Occasionally the contents of the ducts or of a cyst are cheesy or almost solid

FIG. 95.—Chronic mastitis, with formation of cysts containing cheesy material. The breast was removed several years after the last pregnancy. It was infiltrated with eosinophil cells (*Fig.* 78).

in consistency (*Fig.* 95). This material may consist chiefly of enormous numbers of cast-off cells. Occasionally it may be composed almost entirely of fat, and is then probably derived from inspissated milk. A cyst of this nature is known as a galactocele, and is likely to arise in a breast which has attempted to lactate in the presence of a mastitis already well established or of a carcinoma, either condition causing an obstruction of one or more ducts.

I have collected the fluid from an ordinary smooth-walled cyst of the breast and found it to be strongly alkaline to litmus. Professor F. R. Fraser has, however, determined for me that its hydrogen-ion concentration is the same as is found in other body fluids. Investigations which may have some bearing on this problem have recently been recorded by W. Taylor.[15] He has shown that milk contains, in addition to its specific constituents, various nitrogenous extractives, such as amino-acids, urea, uric acid, creatin, and creatinin, all of which are to be regarded as excretory rather than as secretory products, the amounts varying with the degree of their concentration in the

blood. But these observations are made only on the threshold of a gate to further knowledge.

The chemical changes which may take place in these stagnating fluids are, as has been said, more or less unknown; but I feel sure that much will be learnt from the investigation of the chemistry of the secretions and of their effect upon the living cells that are exposed for a longer or a shorter time to their influence.*

Attention has several times been drawn to the analogy between the changes that are found in middle age in the breast and in the prostate,[16] and it is quite possible that chronic mastitis and senile hypertrophy of the prostate are dependent upon similar factors. Light may also be thrown upon abnormal epithelial growth in other organs.

THE RELATION OF MASTITIS TO OTHER NON-MALIGNANT DISEASES OF THE BREAST.

The nomenclature of the various non-malignant affections of the mammary gland at present in use is confusing, and introduces many needless complications. This seems to be due chiefly to a lack of appreciation of the essential continuity of so many of the non-malignant lesions found in the breast, separate names and descriptions having been applied to different phases of a chronic inflammatory condition as they were noticed, so that the student is confronted with a series of lesions, each apparently a clinical entity. Too much attention has been focused on the local manifestations, and too little on the underlying cause. I have already demonstrated the great frequency with which a chronic non-bacterial inflammation is met with in the breast. This may be conveniently referred to as *chronic mastitis*, and to this may be assigned the origin of many of the non-malignant lesions in the breast. It cannot, of course, be held responsible for causing the encapsulated tumours of the breast, such as the fibro-adenomata, which are true adenomata, and are as inexplicable as analogous tumours occurring in any other part of the body.

Starting, therefore, with the chronic mastitis of the text-book, we find that an artificial distinction is made between *lobar* and *lobular* mastitis, between *interstitial* and *parenchymatous* mastitis. These distinctions depend only on the incidental distribution of the most obvious part of the lesion—most obvious, that is, to the fingers of the investigator—or on the kind of reaction, whether fibrous or epithelial, which happens to predominate in any given individual. I have shown that a microscopic dilatation of ducts and acini is an early manifestation of chronic mastitis. As this dilatation progresses, small cystic spaces become isolated, and undergo progressive enlargement owing to the accumulation of fluid secretion. They finally become evident to the touch, and if the lesion is diffuse the condition is called *chronic cystic mastitis*, *sero-cystic disease*, or *cystadenoma of Schimmelbusch*. Often the lesion is more or less localized, and a single large cyst or a group of cysts results.

* Since this was written, further evidence has already appeared in the observations upon living cultures published by A. H. Drew (*Brit. Jour. Exper. Pathol.*, 1923, iv, 46), who has shown that cellular growth is stimulated by the presence of the products of autolysis of cells.

These are described separately under the name of *simple cyst* of the breast, but always the microscope reveals a diffuse condition of chronic mastitis in the neighbourhood of the large and obvious cyst. Often these cysts contain a clear fluid, and the epithelium lining them is reduced to an inconspicuous layer of flattened cells. Such cysts have even been interpreted as dilatations of lymph spaces, under the name of *interacinous cysts*[17] of the breast. Sometimes pronounced epithelial changes occur in these cysts, with the production of papillomatous growths inside their lumen. These are then described under the heading *papilliferous cysts.* If the papillomata happen to have grown chiefly in the large collecting ducts, then a condition known as *duct papilloma* is diagnosed. The presence of a papilloma in the ampullæ of the ducts often gives rise to a clear or blood-stained discharge from the nipple, and this has been interpreted as a clinical condition of some gravity ;[18] but usually it denotes nothing more than this particular manifestation of a chronic mastitis. The extreme degree of papillomatous growth within a cyst in which both cyst and papilloma are of large dimensions has been described under the name *Brodie's tumour*,[19] but this is seldom seen at the present time. A distinction is also made when the contents of the cyst consist chiefly of fat, whether yellow or white in colour. This is formed from inspissated milk, and may result from a 'stagnation mastitis' following lactation, or attempted lactation in the presence of pre-existing mastitis. It is called a *galactocele*, but is not a common condition.

Various other names have been used by different writers. Bloodgood[20] has dubbed the isolated cyst containing clear fluid the 'blue-domed cyst', owing to the appearance it presents when the deep surface is exposed. This writer[21] has also applied the name *senile parenchymatous hypertrophy* to the cystic form of chronic mastitis, but I believe this implies an erroneous interpretation of its origin. He has also classified the different histological appearances seen in chronic mastitis under a great variety of complicated names, some being even self-contradictory, such as 'non-encapsulated cystic adenoma'. This appears to be somewhat unnecessary. In considering chronic mastitis, if the fundamental changes that may be found in epithelial and interstitial tissues are appreciated, then the possible permutations and combinations of the changes may be assumed, and do not need special designations.

Although this section is headed " The relation of chronic mastitis to other non-malignant diseases of the breast ", it is evident that I tend to regard most of the separately-named conditions as manifestations of chronic mastitis rather than as 'other diseases'. I do not, however, want to exaggerate this attempted simplification. There are other conditions in the breast, such as the *diffuse hypertrophy* sometimes seen in young women, which cannot be included in this category, and a number of other lesions, definitely infective or traumatic in origin, which I need not specify here.

THE RELATION OF MASTITIS TO CARCINOMA OF THE BREAST.

The subject of the last section was mainly of academic interest. I now turn to a difficult subject of the greatest clinical and scientific importance. An immense body of literature has been devoted to the discussion of the relation between chronic mastitis and carcinoma of the breast. This seems to

begin with the statement made by Billroth[22] in 1880 that " cancer does not develop in an otherwise normal breast ". It culminates in the evidence published by McCarty[23] in 1915; the changes of chronic mastitis were present in every one of 967 cancerous breasts investigated by him. The opinions of many other writers tend in the same direction, and it is, indeed, an almost universally accepted idea that carcinoma of the breast is preceded by the epithelial changes of chronic mastitis. McCarty claims that in chronic mastitis three distinct histological pictures are to be seen. He distinguishes in the normal acinus of the breast two layers of cells—an inner layer of cubical secreting cells, and an outer layer of 'basket cells', which may be the precursors of the inner layer or only a supporting or nutritional layer. He then describes in chronic mastitis: (1) Hyperplasia of the outer layer, the inner layer intact; (2) Hyperplasia of the outer layer, the inner layer cast off and gone; (3) Hyperplasia of the outer layer, with infiltration of the basal membrane which previously limited the cellular growth.

Thus chronic mastitis has become carcinoma, and the trick is done. It seems simple enough; but this interpretation of the histological appearances has not met with universal acceptance, and the transition cannot yet be taken as proved. More recently Sir Lenthal Cheatle[24] in several valuable contributions has tried to demonstrate the same passage from innocent to malignant proliferation, and has even been so bold as to class under the heading of 'the proemial breast' a mammary gland which shows the epithelial changes previously described. But the cases which he has recorded under this designation appear to be fairly typical of an advanced stage of one variety of chronic mastitis, and it has been pointed out elsewhere[25] that carcinoma is seldom or never seen in diffuse cystic mastitis. The appearances seen in such breasts certainly have been interpreted by some pathologists as actual carcinoma. But that this is erroneous seems to be shown by the clinical history of such cases after operation. All such patients are definitely cured by removal of the breast. Recurrence never takes place, axillary glands are not invaded, and the condition does not behave in any respect as if it were malignant. The absence of true histological infiltration agrees with this. If definite infiltration with epithelial cells can be demonstrated, then cancer must be diagnosed. But the co-existence of innocent and malignant proliferation does not prove that one necessarily precedes the other.

However probable the occurrence of a 'precancerous' condition in chronic mastitis may appear, it is exceedingly difficult to furnish any scientific proof of the change, and this has certainly not yet been done.

It has already been mentioned that a great many writers have recorded their observations on the occurrence of chronic mastitis in cancerous breasts, and that McCarty found the association in every one of 967 breasts. It may, in the face of this, seem futile to refer to my own observations, which have been made on only 25 specimens. But even in this short series I have been struck with one or two facts. In these 25 breasts I have found some evidence of chronic mastitis at a distance from the carcinoma in 20, that is 80 per cent. Of the remaining 5, 3 had been infiltrated throughout with the carcinoma, and one was, in addition, lactating, so that it was not possible to form any opinion as to whether chronic mastitis had been associated with the carcinoma

or not. It seems to me very extraordinary that this same difficulty should not have occurred in McCarty's series of 967 specimens. The remaining 2 specimens of my series showed only a senile atrophy in the outlying parts of the breast. I was not therefore able to confirm the 100 per cent results of some observers.

Another singular fact that forced itself on my attention was that in most cases the breast tissue showed a great increase in epithelial activity close to the advancing edge of the carcinoma, but that this was progressively less the greater the distance from the malignant infiltration. This proliferation, which resembles that seen in the proliferative type of chronic mastitis, looked as if it were in some way connected with the growth of the carcinoma in close proximity. The well-marked degree of infiltration with lymphocytes usually seen at the growing edge of the carcinoma indicated an inflammatory reaction, and strongly suggested that the malignant cell might be influencing other cells at a distance by means of an abnormal and irritating secretion which induced a cellular proliferation similar to that resulting, as I have suggested, from long exposure to secretions in chronic mastitis, though acting very much more rapidly.* If this suggestion were to be substantiated it would explain the apparent association of chronic mastitis with carcinoma in so large a proportion of cases; but it would in no way demonstrate that chronic mastitis is a 'precancerous' condition.

I cannot offer an opinion that would carry any weight as to the precise relation of chronic mastitis to carcinoma. I can only make the suggestion that carcinoma is not necessarily preceded by chronic mastitis, but that both conditions, or either separately, may result from one cause—namely, prolonged exposure to a chemical irritant such as may be present in the stagnating secretions of the breast. There is no reason for supposing that chemical irritation produces exactly the same reaction in the epithelial cells of different individuals. In some the cells appear to be more vulnerable than in others, so that the effects are apparent at an earlier stage, or the cellular proliferation tends to be of a malignant rather than of an innocent type. The factors governing the results are exceedingly obscure, but it is noticeable that the time factor seems to have much the same influence in the incidence both of carcinoma and of chronic mastitis in its severer forms. Thus both are commonest a few years after the child-bearing period in married women, and occur rather earlier in unmarried women. There is a popular belief that to have a large number of children is the best way of avoiding cancer of the breast. This is in agreement with the hypothesis that is put forward here.

The proof that chemical irritation may produce a carcinoma of the breast has recently been furnished by experiments carried out in Tokio.[26] Various forms of tar were injected at regular intervals into the mammary glands of mice. Carcinoma resulted in over 12 per cent of the experiments, so that the mammary gland epithelium has now been shown to respond to this particular chemical irritant in the same way as the epithelium of the skin. This was to be expected, but it has now been established by experimental proof.

---

* This suggestion tends to be confirmed by the experiments upon living cultures published by A. H. Drew (loc. cit.) since the above was written. Malignant tumour cells have been shown to contain a substance which acts as a potent stimulus to cellular proliferation.

THE TREATMENT OF CHRONIC MASTITIS.

In considering the treatment of chronic mastitis I shall not enlarge upon the operative methods of dealing with the more advanced stages of the disease. It is clear that in the present state of knowledge it is wisest to deal radically with the fully-developed type of chronic mastitis with cysts. Complete removal of the gland is necessary, though it may be remembered that, if there is no reason to suspect the presence of a carcinoma, there is no necessity for removing much, or even any, skin, and that a subareolar operation may be carried out without fear of recurrence. This may still be done even though the massive proliferation of epithelial cells is found to be present. If the disease is apparent as a single cyst, or a localized group of cysts, then a local operation is adequate. Some diffuse change will almost certainly be present in the remainder of the breast, but it is always possible that this will not develop further and will not give rise to symptoms of any consequence. The same remarks may be applied to a papilloma of a duct-ampulla or to a galactocele. Evacuation of a simple cyst by aspiration with a syringe is a palliative procedure, which is likely to be only temporary in its effect. A local operation is to be preferred.

The treatment of the earlier stages of chronic mastitis in which pain and tenderness, or merely discomfort, are the chief symptoms, presents a more difficult problem. Often there is a psychological factor in the symptoms, and operation is scarcely ever to be advised. If the trouble is of the intermittent menstrual type, no medical treatment or local application is likely to be of any avail. This type occurs chiefly in young women, and the symptoms may subside of themselves as the patient grows older. Otherwise marriage, followed by pregnancy and lactation, will relieve the patient of her trouble. The more persistent type of chronic mastitis in older patients I have attempted to treat in the way which is logically suggested by the investigations here described. I have first freed the mouths of the ducts in the nipple and opened up their natural outlets by bathing with hot water. Manual pressure combined with a suction bell will then often produce a considerable quantity of the turbid or clear green fluid which is always present in the ducts and dilated acini. The patient is instructed to carry out this treatment systematically, and in a few cases some relief of pain and discomfort has been obtained. But the results are disappointing on the whole. This is not unnatural, for the chronic inflammation has produced fibrosis and other abnormalities in the glandular system, so that efficient drainage of every lobule is no longer possible. The treatment should not be persisted in unless improvement very soon results.

The best results seem to be obtained by the judicious application of X rays. If the proper dosage is applied by an expert, a single treatment will usually have a most satisfactory effect. By this means the secretory activity of the epithelial cells is inhibited or destroyed, and the treatment is again the logical outcome of the interpretation I have put upon the pathology of the disease. The treatment is not new, having often been used empirically in the past, but I have here attempted to put it upon a rational basis.

CONCLUSIONS.

1. The breast is a secreting gland which shows periodical activity from birth to the menopause.

2. The normal, non-lactating breast has no outlet through the nipple for the discharge of its secretions. Secretion must therefore be balanced normally by re-absorption.

3. Chronic mastitis is manifested by dilatation of ducts and acini, accumulation in them of the products of epithelial activity, infiltration with lymphocytes, fibrosis, and epithelial changes. Distribution of all these is very erratic.

4. Chronic mastitis is commonest in women, but occurs also in men. It first appears in the second decade, but is most often seen in the fifth. It appears earlier in single women than in women who have borne children.

5. Chronic mastitis is not bacterial in origin, toxæmic or traumatic, nor is it related to involution changes in the breast.

6. The cause of chronic mastitis is probably to be found in chemical irritation due to stagnating secretions and epithelial débris. This cannot be proved until the chemical changes have been investigated.

7. Chronic mastitis is the underlying cause of many lesions usually described as clinical entities, such as simple cyst, papilliferous cyst, or galactocele.

8. Chronic mastitis, though very often associated with carcinoma, has not been proved to be 'precancerous'. Both may be due to the same cause.

9. Chronic mastitis in its more advanced stages must be treated by operation. At an earlier stage natural drainage may be tried, or X rays may be applied.

REFERENCES.

[1] INGLEBY, H., *Brit. Med. Jour.*, 1919, ii, 631.
[2] SPENSER, E., *Faerie Queene*, II, xii, 78.
[3] FINNEY, M. T., *Keen's Surgery*, 1911, iii, 573.
[4] SCHNELLER, J., *Arch. f. klin. Chir.*, 1922, cxix, 169.
[5] CHEATLE, SIR G. L., *Brit. Med. Jour.*, 1921, i, 895.
[6] LOCKWOOD, C. B., *Cancer of the Breast*, Oxford, 1913, 2.
[7] BAINBRIDGE, W. S., *Amer. Jour. Obst. and Gynecol.*, 1921, i, 465.
[8] BRADLEY, H. C., *Physiol. Reviews*, 1922, ii, 415.
[9] BERGEL, S., *Die Lymphocytose*, Berlin, 1921.
[10] BERTELS, A., *Deut. Zeits. f. Chir.*, 1913, cxxiv.
[11] LUKOWSKY, A., *Deut. Zeits. f. Chir.*, 1921, clxvii, 81.
[12] BINNIE, *Regional Surgery*, 1917, i, 584.
[13] CREIGHTON, C., *Contributions to the Physiology and Pathology of the Breast*, London, 1878.
[14] SHAKESPEARE, *As You Like It*, II, vi.
[15] TAYLOR, W., *Biochem. Jour.*, 1922, xvi, 611.
[16] BLOODGOOD, J. C., *Surg. Gynecol. and Obst.*, 1906, iii, 721.
PAUL, F. T., *Lancet*, 1910, ii, 294.
[17] ROSE and CARLESS, *Manual of Surgery*, London, 1920, 1051.
[18] LEWIS, E. C., *Lancet*, 1922, i, 1093.
DICKINSON, G. K., *Amer. Jour. Obst. and Gynecol.*, 1922, iii, 31.
[19] POWER, SIR D'ARCY, "Brodie's Tumour and Brodie's Abscess", *Brit. Jour. Surg.*, 1922, ix, 334.
[20] BLOODGOOD, J. C., *Arch. of Surg.*, 1921, iii, 445.
[21] BLOODGOOD, J. C., *Surg. Gynecol. and Obst.*, 1906, iii, 721.
[22] BILLROTH, R., *Deut. Chir.*, 1880, xli, 110.
[23] MCCARTY, W. C., *Collected Papers of the Mayo Clinic*, 1915, vii, 918.
[24] CHEATLE, SIR G. L., *Brit. Med. Jour.*, 1922, i, 869; *Brit. Jour. Surg.*, 1921, ix, 235.
[25] BLOODGOOD, J. C., *Arch. of Surg.*, 1921, iii, 445.
[26] YAMAGWA, K., and MURAYAMA, K., *Jap. Med. World*, Tokio, 1922, ii, 337.

CONGENITAL ILEOCÆCAL CYSTS.

By H. F. MacAULEY, Dublin.

There is a group of cases of intestinal cysts, with a very distinct pathology and definite symptomatology, which merits a place in surgical literature. Descriptions of individual cases of this group have appeared from time to time in the course of many years, but no attempt, so far as I am aware, has been made to collect these cases; this is unfortunate, as they form a group not only distinct and characteristic, but also of more than passing surgical interest. In this paper I add one more to their number, and propose to collect and give a short account of all the other cases in such literature as is available.

The following is the history and its denouement in regard to my own case:—

A baby girl of six months was brought to hospital with typical symptoms and signs of intussusception, and the usual 'tumour' was palpated, extending into the descending colon. Operation was undertaken immediately after admission, which was exactly twenty-four hours after the initial spasm of pain and associated vomiting was first noticed.

Fig. 96.—Showing the cyst projecting into the cæcum. (*Natural size.*)

It is noteworthy that the past history of the child was entirely negative as regards any gastro-intestinal complaint, with the exception of a short attack of 'colic' when the baby was six weeks old. The physique of the child, as is customary in cases of intussusception, was in every way excellent, and the baby had been nursed by the mother.

At operation, reduction even in its early stages was carried out with unusual difficulty for a case of this duration. When the 'tumour' had all been reduced except its terminal portion, the cæcum and adjacent ileum were brought outside the abdomen so that the final manipulation and appraisement of reduction could be carried out in full view. It was then that the unusual nature of the case was first noticed. To the eye it appeared that reduction was complete, for there was no fossa or peritoneal infolding around the entrance of what was the intussusception. Yet a hard knob-like structure could still be palpated in the interior of the cæcum on the site of the ileocæcal valve—apparently a portion of still unreduced bowel—and a finger could not be invaginated through the cæcal wall into the ileal lumen. A few further

gentle attempts were made to reduce the final mass, but without success. The gut was then resected, side-to-side anastomosis carried out, and the child returned to bed. Six hours later, however, a fatal issue ensued, due to post-operative shock.

Specimen.—The resected gut was examined, and the explanation of the presence of a mass in the ileum while reduction was apparently complete was forthcoming. On incising the ileum and cæcum a very definite, thick-walled, tense cyst was seen, extruded through the ileocæcal valve.

The specimen was despatched to Professor Sir Arthur Keith, to whom I am indebted for the report, and is now in the Museum of the Royal College of Surgeons of England. The appended drawing (*Fig.* 96) gives a good idea of the actual specimen.

> **Report.—A congenital cyst at the ileocæcal junction, which became intussuscepted within the cæcum at the ileocæcal angle. The cyst wall has the same coats as the small intestine, with interior quite cut off from the lumen of the bowel, and yet lined with ordinary mucous membrane. I regard such as diverticular cysts arising by outgrowth from the embryonic bowel, but know no particular reason as to why they should arise at the ileocæcal angle.—(*Prof. Sir A. Keith.*)**

OTHER CASES IN THE LITERATURE.

The earliest description of a similar case was made by Fränkel[1] in 1882. Unfortunately a microscopic examination was not made, but the autopsy findings and the age of the child are undoubted evidence that this was a case of congenital ileocæcal cyst. At the autopsy on the child, who died on the third day after birth with symptoms of intestinal obstruction, a cystic tumour was found at the termination of the ileum. The cyst was round, 2½ cm. in diameter, and projected both into the ileum and cæcum, and was only apparent after incising the bowel wall.

I have rejected an earlier case described by Conant[2] in 1856, as the evidence is insufficient for its inclusion. In this case a cystic tumour containing a thick, creamy, semi-solid substance was found at autopsy in a male subject; it was attached to the ileum at its junction with the cæcum. This may have been a broken-down tuberculous lymph gland.

However, a case described by Sainsbury[3] in 1886 is in a different category, and quite possibly truly belongs to the group under review, though the author regarded it as of different nature. At autopsy on a girl of 11 years who had died of typhoid, a large cyst was found in the interior of the colon, just above the ileocæcal valve orifice. The cyst was entirely cut off from the bowel lumen; its wall was formed by the muscularis mucosæ of the intestine, and in parts a distinct muscular wall was present; the interior of the cyst was smooth and apparently covered with a serous membrane. The author inclined to the view that the cyst arose from a sequestrated portion of peritoneum, which gradually became distended by the exudation of fluid. He also suggested the possibility of the case being a mucous retention cyst or an enterocystoma. It is difficult to classify the case; but its position, the muscular structure of its wall, and the lining—which not improbably was ordinary

intestinal epithelium flattened by pressure—makes the diagnosis of enterocystoma very possible.

As a contrast to these doubtful cases, a very definite one was recorded by Sprengel,[4] which is very similar to my own case. It occurred in a girl of 15 years, and was the occasion of an intussusception. The cyst was in the wall of the ileum, quite close to the ileocæcal valve. At operation the intussusception was irreducible, and on examining the resected gut the cyst was found. The cyst wall was typical, an exact replica of the structure of the intestinal wall. This girl had suffered from periodical attacks of pain and vomiting since she was 4 years old. Probably the cyst was present since birth, but only became sufficiently large at the fourth year to encroach on the bowel lumen.

Two years later Hedinger[5] described a case which occurred in a boy of 4 years. Here the cyst was very large, and filled a great part of the abdomen, though it appeared to have originated in the ileocæcal region. Portions of the cyst wall showed typical intestinal structure, all the layers being reproduced, whereas in other parts the wall was only represented by connective tissue. The cyst was filled with lymph. The large size and contents of this cyst were apparently due to the rupture of a lymph vessel in the wall of the enlarging cyst. This child had been suffering from abdominal troubles for a long time previous to examination.

Ayer[6] described a case in a man of 23 years, who was operated on for supposed appendicitis, with a severe attack of pain, vomiting, and constipation. These symptoms were present in a milder degree for years before operation. The case turned out to be a thick-walled cyst, about the size of a duck's egg, in the cæcum. Here, as so often is the case—even with the abdomen opened—the diagnosis was in doubt, and the case was thought at first to be an intussusception. Then the cæcum was incised, and the cyst found overhanging the ileocæcal valve. There was no microscopic examination, but macroscopically the thick-walled cyst was apparently lined by mucous membrane. It is interesting to note that there was discovered at the site of the attachment of the cyst in this case a funnel-shaped diverticulum, which extended for about two inches between the layers of the mesentery and parallel with the ileum. The abnormal diverticulum in this case I would think due to the fact that the entire cyst was originally situated at the ileocæcal angle, but that owing to growth and intestinal movements the part of the cyst wall near the bowel was extruded into it, and hence the cyst became hourglass-shaped. Despite the absence of microscopic evidence, I believe this case is a genuine one of ileocæcal enterocyst—though the author, apparently on account of Dowd's[7] teaching, attributed the case on slender evidence to a rest of the Wolffian body.

While in the cases previously described the cyst originated as a diverticulum from the small intestine, and was lined by mucous membrane corresponding to that part of the intestinal tract, in a case described by Krogius[8] the cyst arose as a diverticulum of the large bowel. The patient was a child of 2 months, and was operated on for intussusception. Here the site was the same, the ileocæcal angle; a well-developed muscular layer was also present; the epithelium was cylindrical, but contained numerous tubular glands. The

lumen of the bowel was narrowed by the growth, which was as large as a pigeon's egg.

In this category it is interesting to recall a case operated on by Baldwin,[9] where a diverticulum, which contained all the coats of normal intestine, was found at operation projecting from the cæcum at a point directly opposite to the entrance of the ileum.

Blackader[10] described a cyst which occurred exactly at the site of Baldwin's diverticulum. The usual findings appeared : a child of 10 weeks, in this case a boy, was brought to operation for intussusception ; this turned out to be irreducible, and the gut was then resected. Only when the incised intestine was examined did the true condition become manifest, a tense unilocular cyst being found situated in the wall of the cæcum, opposite the ileocæcal valve, and extending over and completely obstructing that orifice. The epithelium was columnar-celled and contained tubular glands, and outside this the other intestinal layers were present. The case was diagnosed as retention cyst ; but it was undoubtedly a case of enterocystoma.

The three following cases were all described in fairly recent English medical literature, and are all typical examples of the group of cases under discussion.

The first is described by Turner and Tipping.[11] It occurred in a child of 4 months. The child had been in good health until a week before admission. During that week there were vague symptoms of irritability, followed on the day of admission by those of acute intussusception. Laparatomy was performed, and a tense cyst about one inch in diameter, situated in the ileocæcal angle, was found to encroach on the lumen of both the ileum and cæcum. The cyst wall was incised between the layers of the mesentery, and much of the wall removed ; the cut ends were stitched to the parietal peritoneum. A section made from the excised wall showed the structure of small intestine, with a lining of typical intestinal mucous membrane.

The second case was that of Ball,[12] and occurred in a child of 3 months. Symptoms of intestinal obstruction were present for two days previously. Again, even after laparotomy—as occurred in practically every case recorded —the condition was thought to be an irreducible intussusception. Here the colon was incised, and a tensely distended cyst was seen to project from the wall of the cæcum and block the ileocæcal orifice. Resection was then carried out. The inner layer of this cyst was formed of flattened epithelium ; outside that was a layer composed of loose fibrous tissue and non-striped muscle.

The third case showed the cyst projected into the ileum, just on the ileal surface of the ileocæcal valve. The cyst, in the words of the authors, Bolton and Lawrence,[13] " lay in the mesenteric aspect of the ileum between the layers of the mesentery, and the terminal ileum coursed over it." The structure of the wall was a very complete reproduction of the intestine, even to the two distinct layers of smooth musculature. This specimen occurred in a baby girl of 3 months, who had suffered from gastro-intestinal complaints from birth, and was found at autopsy.

Only one other example remains, and it is that published and illustrated by Keith[14]. It possesses some features distinct from others. At autopsy in a new-born child a small ileocæcal cyst was found. In this instance the

cyst lay in the mesentery removed a little distance from both ileum and cæcum. In other respects the case was typical, and the cyst wall reproduced the intestinal structure. The cyst was small, and is interesting as occurring in such a young child, showing what is probably the original site of these cysts before they have to change their position as a result of growth expansion.

SUMMARY.

The group, as is apparent, is a very composite one.

1. The site of the cysts is very constant at the ileocæcal angle.

2. The symptoms are those of obstruction, and appear most frequently in the first six months after birth; but occasionally, with a slow expansion of the cyst and a favourable position, may be delayed until adult life.

3. In childhood the cases are mostly frequently diagnosed intussusception, even after the abdominal wall is opened, and not infrequently an intussusception, as in my own case, has been caused by the cyst.

The reason the ileocæcal angle should be a favourite place for these enterogenous cysts is entirely obscure. Formerly, practically all intestinal cysts from the cæcum to the jejunum, whether on the convex or the concave surface of the intestinal loop, were attributed to Meckel's diverticulum; but this explanation is scarcely tenable for the latter, especially when situated as low as the ileocæcal angle.

My thanks are due to Professor Keith for his report, and for help in facilitating my access to some of the literature.

REFERENCES.

[1] FRANKEL, *Virchow's Arch.*, 1882, lxxxvii, 275.
[2] CONANT, *N. Y. Jour. Med. Sci.*, xvi, 82.
[3] SAINSBURY, *Trans. Pathol. Soc. Lond.*, 1887, xxxviii, 146.
[4] SPRENGEL, *Arch. f. klin. Chir.*, 1900, lxi, 1032.
[5] HEDINGER, *Virchow's Arch.*, 1902, clxviii, 29.
[6] AYER, *Amer. Jour. Med. Sci.*, 1906, cxxxi, 89.
[7] DOWD, *Ann. of Surg.*, xxxii, 515.
[8] KROGIUS, *Zeits. f. klin. Med.*, 1903, xlix, 53.
[9] BALDWIN, *Med. Record*, 1912, lxxxi, 991.
[10] BLACKADER, *Trans. Amer. Pediat. Assoc.*, Chicago, 1913, xxv, 296.
[11] TURNER and TIPPING, *Proc. Roy. Soc. Med.* (Sect. Dis. of Children), 1913–14, vii, 29.
[12] BALL, *Brit. Jour. Child. Dis.*, 1914, ii, 259.
[13] BOLTON and LAWRENCE, *Brit. Med. Jour.*, 1916, ii, 248.
[14] KEITH, *Brit. Jour. Surg.*, 1921, viii, 452.

PATHOLOGICAL REMARKS ON SARCOMA OF THE LONG BONES.

By Professor S. G. SHATTOCK, F.R.S.

The following observations are confined to a few selected topics in relation to the above subject.

Nomenclature.—

Giant-celled Tumours of Bone.—The innocent giant-celled tumour of bone is now generally known as a myeloma. A still better name, I have long thought, would be 'giant-celled myeloma', implying that the elements of the bone-marrow concerned are particularly the multinucleated osteoclasts, and not the myelocytes as in some forms of myelomatosis.

But at once we are confronted with the difficulty that some of the giant-celled tumours of bone are distinctly malignant and produce metastasis—a fact fully recognized by Sir Henry Butlin. How are such discrepancies to be reconciled ?

The origin of giant-celled tumours in soft parts quite independently of bone is sometimes ignored. There are in the collection of general pathology in the Royal College of Surgeons, London, two examples of this. One is a tumour growing in the adductor muscles of the thigh ; it is as large as the fist, and recurred after free removal. The microscopic sections of the neoplasm present in some places the features of spindle-celled sarcoma, and in others of myxosarcoma. Throughout the growth there are distributed considerable numbers of multinucleated giant cells. The second is a still larger primary tumour growing in the mesentery. It was removed together with a loop of the contiguous intestine ; this tumour also recurred, and on subsequent exploration of the abdomen was found to have become insusceptible of further operation. Microscopic examination shows it to be spindle-celled, arising in lymphatic glands. Intermingled with the other elements there are large numbers of multinucleated giant cells.

Under the term myeloma, the simple giant-celled tumour of osteoclasts, and the true giant-celled sarcoma, have, it would appear, been included.

Endothelioma.—This name is by some used in the most unmethodical manner : it should be limited to benign tumours arising from the endothelium of lymph spaces or blood-vessels. It is not our practice to give a common name to simple and malignant tumours; yet without compunction we accept such terms as benign and malignant endothelioma. If an endothelioma is malignant from the first, or becomes so secondarily, the best name for it, by far, is endothelial sarcoma : this is more illuminating than the older names of plexiform and alveolar sarcoma.

Infection of Lymphatic Glands.—

There are two forms of sarcoma especially adapted to infect lymphatic glands secondarily—the lymphosarcoma and the endothelial sarcoma, of bone or elsewhere; the reason being that the tumour cells from the beginning lie in free connection with lymph radicles. Of lymphosarcoma of a long bone—i.e., a sarcoma of lymphatic tissue—I have seen but a single example: of the lower end of the femur. Of endothelial sarcoma, the most marked case I recall was an intact primary tumour infiltrating and replacing the head of the tibia in a man of 47. The limb was amputated. The growth when divided was highly alveolar to the naked eye, its meshes being filled with large, flat, polygonal cells. Death occurred from exhaustion. On dissection, the popliteal and femoral (deep inguinal) lymphatic glands were found enlarged from secondary disease on the side of the primary tumour.

By reason of the necessary relationship between the tumour cells and the lymphatics, it might at first be doubted if an endothelioma can be anything else than malignant. But in this connection we have to remember the essential biological difference that exists between the cells of benign and of malignant growths, as shown in spontaneously appearing tumours in mice: the benign tumour—e.g., adenoma of the mamma—does not admit of being grafted, either into the same mouse or into others; whereas the malignant tumour—e.g., a squamous-celled carcinoma of the vulva—can be successfully grafted on the same or on other mice.

It is not the simple transference of elements, then, that suffices to produce metastatic growth; there is a profound difference in the biology of the simple and the malignant cell: when we know in what this consists we shall know the pathogenesis of malignant disease. Not the least tantalizing thing in regard to malignant growth is that one can see so clearly how it starts and how it progresses, without being able in the least to explain the why.

The Removal of Portions of Sarcoma for Histological Diagnosis.—

This practice is at present widely condemned by surgeons, and yet almost as widely resorted to. The removal is obviously accompanied with the opening of vessels and the displacement of tumour cells into or over their divided ends. The grossest instance of dissemination produced by exploration of a bone sarcoma of which I personally know was that of an osteoid tumour which surrounded the lower end of the shaft of the radius in a young man. The swelling was thought to be possibly syphilitic, or due to some form of necrosis accompanied with the formation of an involucrum. It was cut into and explored. A week afterwards an enlarged gland was felt at the elbow and another in the axilla; blood-stained fluid was withdrawn from the pleura, and within two months of the exploration death had occurred with metastases in both lungs.

Dry heat, however, is, I find, a satisfactory means of tissue fixation. Excellent histological sections may be prepared from the thoroughly cooked muscle of roast mutton, or from cubes cut out from kidney thoroughly cooked by frying. Heat is, after all, only a mode of coagulating the cell proteins, and fixing the tissue. In microscopic sections of roast mutton, stained with logwood and eosin, the striated muscle is well dyed with the latter, and preserves its double striation, the nuclei of the sarcolemma are perfectly stained with

the hæmatoxylin, and so are those of the cells of the walls of the arterioles and of the general connective tissue: even 'Miescher's cylinders' in some of the muscle fibres are quite well displayed; and so in the kidney, *mutatis mutandis*. Then why not evade the danger by trying heat?

With the technical aid of Messrs. Allen and Hanburys I have devised an electrically heated borer to remove a cylinder of tissue, with a view of coagulating and killing it as the instrument is pressed forwards. The borer is heated by an 'element' contained in its wall (which is double), the coil of which returns on itself and is connected at each end with a leading wire. The borer works in an outer double tube (the space in which is packed with asbestos) fitted to a centrally bored box-wood handle, and is furnished with a plunger to push out the included cylinder of coagulated tissue from its cutting, penetrating end.

In using any such boring instrument, the essential indication would be to penetrate the tumour very slowly, in order to coagulate and kill not only the tissue for microscopic examination within the borer, but also that immediately around it. One technical difficulty to overcome is to reduce the diameter of the borer to the required size: there seems to be no way of heating it by any mode of conduction from the proximal end; the inclusion of a coil in its wall involves some thickness, and corresponding increase in diameter; a borer not exceeding a quarter of an inch would be the maximum desirable. Another technical difficulty is the detachment of the cylinder of tissue: the coagulation would extend beyond the cutting edge in front; but how to disengage the cylinder of coagulated tissue without tearing through the uncoagulated?

The Rarity with which the Growth of Sarcoma follows Fracture of the Long Bones.—

This is another subject worth reflection. I am personally acquainted with only one case. This was of a healthy policeman who was pushed violently against an iron railing and fractured the shaft of the humerus near its middle. The fracture healed normally and the splints were removed. A month and a half afterwards the patient noticed a nodule for the first time about the site of injury; this increased in size, and was put down as excessive callus. As it continued to increase, sarcoma was diagnosed and the limb was amputated through the shoulder-joint. On dissection, a large tumour was found surrounding the site of fracture. And it is interesting to note that the tumour contained a conspicuous amount of cartilage, as though the cartilage of the callus had participated in the production of the chondrosarcoma. Death occurred two years later with pulmonary metastasis.

It is not that the fracture through the shaft of a long bone occurs through comparatively inert tissue—the adipose medulla and compact wall. What is commoner than transverse fracture of the patella? Yet the growth of sarcoma as a sequence is unknown. It is significant that the same is true of bone tuberculosis; it is not set a-going by fracture; it may be by contusion. The greater injury leads to the greater reaction, and to the local immunization by cells and body fluids against an extraneous factor.

Pathogenesis of Malignant Disease.—

The foregoing considerations lead me to offer an hypothesis of the pathogenesis of human malignant disease, which comprises three elements: (1) That the agent is a filter-passing or ultramicroscopic virus; (2) That this is not a parasite, but is symbiotic, adsorbed to the cell-nucleus and cytoplasm—like a dye to filter paper; (3) That for the symbiosis the cell must be prepared. The preparation takes place usually by chronic inflammation—the great precursor of malignant disease.

Singly, either the first or third factor is harmless so far as malignant growth is concerned. All three acting together, the biology of the cell is fundamentally changed; it is rendered independent of the rest of the body, which it continues to invade, and ultimately leads to the death of the host of which it originally formed a normal part.

One is reminded of Paget's doctrine of the constitutional nature of malignant disease. Interpreted in a concrete form on such an hypothesis, that which is "transmitted" and is "constitutional" would be the ultramicroscopic virus, from the parent to the offspring, without producing any organic lesion in the unprepared placenta, or any necessarily in the offspring; and even being passed on to a further generation until the prepared spot arises.

That what is transferred is not the malignant cell is clear from the fact that in cancerous stock the disease does not necessarily appear in the same organ, nor is it of the same histological kind: indeed, sarcoma may be intercalated amongst carcinomata. The filter-passing virus itself is indifferent or polyvalent: it is the prepared cell that determines the histological form of the disease. This is congruous, too, with the fact that independent and histologically different carcinomatous tumours may coexist in the same subject; or sarcoma may coexist with carcinoma; nay, they may coexist in the same organ, as where a sarcoma of the myometrium grows alongside a carcinoma of the uterine mucosa.

The adsorption or symbiosis of a living ultramicroscopic virus with the cell may be responsible likewise for the abnormal mitosis so conspicuous in the cells of malignant growths; the normal division of the cell could hardly be else than upset under such circumstances.

THE NOMENCLATURE OF DISEASED STATES CAUSED BY CERTAIN VESTIGIAL STRUCTURES IN THE NECK.

By J. ERNEST FRAZER, London.

This is an effort to induce British clinicians to abandon certain terminologies which are inaccurate, misleading, and not sufficiently comprehensive, and to substitute for them a nomenclature or classification which has, at any rate, the virtue of being in accord with embryological facts and possibilities. The hoary legends of our youth still linger in corners of text-books of deservedly high reputation, but the pathetic interest with which we see them ought not to keep us from admitting the possibility of improving on them.

The particular conditions about which I am concerned at present are those which are lumped together under the distinguishing term 'branchial' or 'branchiogenetic'. These terms have been in use unworthily for many years past. 'Branchial cyst' is a common expression in the mouth of the student, and some say that 'branchial carcinoma' is not excessively rare in the neck of a patient.

It must be stated here at once that the question whether such a growth or such a cyst exists or does not exist is not of any importance at all from the present point of view: we are not concerned with that, but only with the provision of a better class-name under which, if it occurs, it would fit naturally, and which it would not require if it does not occur. As a matter of fact, certain developmental vestiges in the neck undoubtedly give rise at times to certain pathological formations; so the necessity exists for an accurate and comprehensive nomenclature.

The objection to the use of the word 'branchial' in this connection lies in the fact that these things are not branchial in the strict sense, have nothing to do with branchiæ, and in many cases are outside the region which might be homologized with the pharyngeal arches that carry gills in the lowest vertebrates. Thus, even if the word be extended to take in the gill-bearing regions, it would still be inadequate and erroneous.

A better conception of the several conditions of this kind that may occur in the neck might surely be obtained by a terminology that puts them in a class with all other diseased vestigial structures in the body, and allows of necessary subdivision and sub-classification, enabling it to include all the possible pathological variations of these structures, in any part.

It is common knowledge that the pharynx of the human embryo possesses in its floor a series of *visceral arches*, with intervening *visceral grooves*—not clefts, as is often wrongly stated. Now these arches do not correspond to the branchial arches in fishes: the *third visceral* arch can be homologized with the *first branchial*, but the two visceral arches in front of this are not properly branchial. Many of the 'branchiogenetic' formations occur in front of the

region which really corresponds to the gill-bearing region of primitive fishes. Moreover, many others are really ectodermal in their origin and—for those who have a predilection for fishy nomenclature—might with a certain amount of propriety be termed 'opercular' or 'sub-opercular'. But why do we in these cases leave the lines of classification that usually suffice in pathological matters, and plunge into the sea after some primeval fish to find a label? These formations are not atavistic, nor, it may be said, do they represent, as they come before the clinician, any condition ever present in any normal animal: they are pathological changes occurring in vestiges of structures normally present in the human embryo, and their label ought to show this. The human embryo never has and never had branchiæ: morphologists believe that the visceral arches and grooves mark a phylogenetic memory of a branchiate stage in evolution, but the individual who possessed those appendages in a bygone time was not human, but at best a tailed amphibian, or at a lower level a sort of mud-eel.

The objection that the 'branchial' label is inadequate is, however, of more direct force than that of its irrelevant opportunism when one is seeking to obtain a generally and universally applicable classification. It would, perhaps, make the matter clearer if a short summary were given of the embryological facts concerned. Disregarding the complexities of the fourth pouch, we can say that each of the four visceral grooves—which lie behind their numbered arches—ends in a deep *lateral pouch* which is close to the surface, its lining entoderm being in fact in contact with the surface ectoderm where this lies at the bottom of a corresponding *external groove*. The lateral pouches have *dorsal* and *ventral angles*, and the surface of contact with the ectoderm extends properly from one angle to the other. In the case of the first pouch, however, there is no definite lower angle, and the contact is only with the upturned dorsal angle. In the case of the second lateral pouch the distance between the angles increases rapidly, the upper angle and the pouch below it lose their contact with the ectoderm, and this is then only found in the region of the ventral angle: the upper or dorsal angle remains permanently in the middle ear, and the lower or ventral angle is placed in the tonsillar fossa. As the neck thickens, the 4th, 3rd, and 2nd (ventral) angles retire from the general surface, but carry with them their external contacts, in this way: the external grooves corresponding to these pouches, with the external arches between these grooves, all lined by ectoderm, are covered in by a hood or fold which grows over them from the dorsal aspect, extending from the 2nd arch in front to the pericardial region behind. Thus a recess lined by ectoderm is formed, at the bottom of which are the external arches and the external grooves which are connected with the internal pouches. This recess is the *cervical* or *precervical sinus*. It is quickly covered in, its opening on the surface is closed, and it apparently disappears: it would be better, perhaps, to say that, like these other remnants, it is no longer to be recognized, and it will facilitate comprehension of the conditions if we imagine it still persisting in some obscure form. Then, as the neck grows, the persisting sac will be drawn away from its close relation to the immediate wall of the pharynx, and its ectodermal contacts with the pouches will be drawn out into long cell-strands, which may contain a lumen, and can be termed *external pharyngeal ducts*, in contrast

with a similar but much less extensive—in human embryos—drawing out of the entodermal pouches, which can be called *internal pharyngeal ducts.* Thus, if this system remained, there would be three *internal* ducts drawn out from the pharynx at points corresponding to the developmental pouches, and these ducts would be continuous through a solid intermedium (the closing plate) with three *external* ducts, which would converge on a common cavity, the *cervical sinus.* The connection of this sinus with the surface is drawn out into a narrow tract, and, as a matter of fact, the external duct running from the region of the second pouch opens into this tract and not into the sinus,

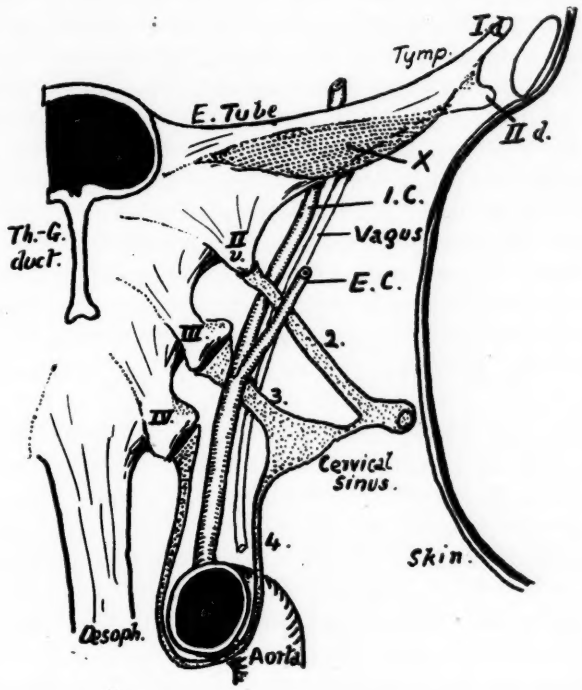


FIG. 97.—Schematic figure to show the vestigial structures in the neck, with their relations to main arteries and nerves. The epithelial bodies are not represented.

I d, II d, Dorsal angles of 1st and 2nd lateral pouches. *II v, III, IV,* 'Internal pharyngeal ducts' derived from ventral angles of 2nd, 3rd, and 4th lateral pouches. *2 3. 4,* 'External pharyngeal ducts' derived from 2nd, 3rd, and 4th external grooves. *X,* Layer of entodermal cells cut off from lower part of Eustachian tube.

(*Reproduced from a drawing by the Author.*)

which therefore has only the external ducts from the 3rd and 4th pouch regions running into its deep aspect. A scheme of the arrangement is given in *Fig.* 97.

There seems to be no reason why any of these various and several structures should enlarge, and also no reason why any one of them should enlarge more than another. Mr. Hamilton Bailey, in his very interesting paper on "The Clinical Aspects of Branchial Cysts" in the April number of this JOURNAL, gives four types of the condition. His first type is one that I would feel disposed to refer to the elongated tract leading to the sinus, or to the 2nd external duct which runs into this tract. His second type seems to

point to enlargement of the sinus itself. The third should be referred, I think, to enlargement of the 2nd external duct, perhaps with more or less enlargement also of the sinus. I may remark in this connection that Mr. Bailey, in quoting from me in this matter, has gathered a wrong impression of my statement regarding the relations to vessels and nerves: if he refers again to the paper he mentions, he will find that I was speaking of 3rd pouch connections only, and am not in any way at variance with clinical experience. These relations will be considered later. The fourth type is a very interesting one to the embryologist, for it may be the remnant of an internal duct, or possibly derived from one of the 'epithelial bodies' which are found in association with the pouches; or it is possibly, in spite of its epithelium, an ectodermal derivative, or one associated with the closing membrane.

These readings of the types given by Mr. Hamilton Bailey are, of course, merely pious opinions of my own. There is only one way of settling the origin of any particular instance of an enlarged vestige, and that is by detailed anatomical examination with reference to relations, followed after removal by microscopic investigation. The question of anatomical relations has to do particularly with the connections with the pharynx, and does not apply so much to derivatives of the sinus placed more superficially: the three pouches and their corresponding external ducts bear definite and distinct relations to the main vascular and nervous structures, and *these relations, if the vessels are normal, are absolutely fixed and certain.* The primary relations are shown in *Fig.* 97.

The 2nd external duct passes between the two carotids and in front of the vagus. The 3rd goes behind the common or internal carotid and in front of the vagus. The 4th is drawn down by the arch of the aorta on the left side, and by the subclavian on the right. These relations are facts, not theories, and are absolutely fixed: if a duct goes behind the main artery and in front of the vagus, it is a remnant of the 3rd duct; but if it does not do so it is not a remnant of this duct. Similarly, the remnant of the 2nd goes between the two carotids to reach the pharynx. Any departure from this arrangement is not possible, if the vessels are normal. Whether or not any particular one of these structures may be enlarged is, as already stated, beside the point; but it may be said that (so far as I know) the only one that has been recognized with certainty is the 2nd; so far as the 4th is concerned, its course would seem to militate against its survival in any dangerous form. The cervical sinus is superficial to the vessels, and its opening is in front of the sternomastoid, drawn down to a low level in the neck. *Fig.* 97 shows how this opening may lead straight into a 2nd duct without necessarily involving the proper sinus at all. If the closing plate were perforated, it is conceivable that any of these ducts, if patent, might lead into the pharynx: the site of the 2nd pouch is at the tonsil, the 3rd at the pyriform fossa, and the 4th at the lower end of the pharynx. Of these, again, a perforation has only been certainly recognized in the 2nd, but it is quite possible that it might exist without demonstration in the 3rd. The ectodermal derivatives might be expected to show a stratified cell-lining, and a columnar-celled layer would probably exist in any entodermal prolongation. Finally, there develops in some lower mammals a 'superficial thymus' in association with the ectoderm of the sinus, and it may

be that the lymphadenoid tissue in the walls of cysts of the sinus (presumably) is really thymic; I have examined some sections of such cysts in which the thymic structure was more than strongly suggested and what looked like badly formed Hassall's corpuscles were distributed through the tissue.

The vestigial structures in this region, then, as shown in the diagram, include entodermal processes of the 2nd, 3rd, and 4th pouches, ectodermal ducts associated with the same pouches, and a cervical sinus with its drawn-out channel of aperture. Mention has been made of other structures connected with these pouches, derived from the 'epithelial bodies' of the pouches: these may possibly form small cysts—associated with the deep aspect of the thyroid gland, or with the parathyroids, or in the upper part of the thorax—which should have their proper place in a complete nomenclature, but they are not represented in the diagram. Certain other structures are shown, however, which should also be included in any classification of vestiges here. One is the thyroglossal duct, about which it is not necessary to say more, as its occasional pathological persistence is well known. The other, labelled *X* in the diagram, is a double layer of entodermal cells which are cut off from the lower aspect of the Eustachian tube. I have described the occurrence of this in the third month, and it is the result of the same process which has separated the dorsal and ventral angles of the 2nd pouch: a forward growth from the 3rd arch destroys this portion of the 2nd pouch and comes up against the 1st groove, which is caught between it and the 1st arch. Morphologically the entodermal part thus caught may represent the lower angle and internal duct of the 1st pouch, and the result is that the first groove, in the tubal region, is wiped out of practical existence, for the opposed layers of entoderm seem quickly to disappear. But the possibility of their persistence is always there: even if it has never occurred up to now, it may happen to-morrow, but the only evidence against its occurrence is purely negative. Hence, from the point of view of this paper, it must be included among the vestigial possibilities. If a cyst of such a vestige were present, it would lie below the tube, behind (at any rate in part) the tensor palati, and in front of the carotid and stylopharyngeus; and if by any chance it opened into the pharynx, it would do so through the sinus of Morgagni.

All these various potentialities for pathological activities, to my mind, call for classification under proper headings. The label 'branchial' only touches indirectly a small part of one of them, is doubtfully applied to another part, has really nothing to do with the rest of it, and is altogether lacking in association with the other vestiges. Even if one submitted to a piscine terminology for a human condition, that would leave the other conditions to be labelled separately. Surely it would be more correct to include all signs of pathological processes in such remnants under the general term *vestigial*: in everyday use, in reference to some particular case, the qualifying term 'cervical', 'abdominal', or whatever it might be, would be dropped as unnecessary. Further subdivision would give us such distinguishing labels as might be convenient: terms such as median and lateral, superficial and deep, ectodermal and entodermal, or whatever word might be suitable, would aid in distinguishing any particular condition among the class of vestigial pathological states. Thus, a persistent and open canal of His would be properly

described as a median or paramedian vestigial entodermal sinus, although in everyday use the labels of 'canal of His' or 'thyroglossal duct' would probably be preferred as less cumbrous, and there are no objections, other than general ones, to their use; the common 'branchial cyst' in the neck would become a lateral vestigial cyst, ectodermal or entodermal as the case might be, and could be more particularly distinguished if its developmental value admitted of definite demonstration: a cystic distention of a persistent remnant of the first groove would be a right or left sub-tubal vestigial (entodermal) cyst; the same idea in terminology would come in, with descriptive accuracy, in fistulæ or carcinoma or in any other condition associated with vestigial structures.

I am afraid that I have written somewhat at length on what may be considered a matter of relatively small importance; but it seems to me that improper labelling is a fruitful source of mistaken ideas and a hindrance to a proper conception of the underlying facts, wherever it is employed. I have tried to show that the word 'branchial' is inapplicable to most of the cases in which it is used, definitely wrong in some of them, and of doubtful value in the others. I confess that I would like to see the word completely dropped from all writings on human anatomy or embryology. A proper nomenclature should include under one heading and in one class the several affections which have their origin from vestigial persistence, in whatever part of the body they may occur, and should allow of rational sub-classification within this class.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD.

PROFESSOR PUTTI AND THE ISTITUTO ORTOPEDICO RIZZOLI AT BOLOGNA.

THE Orthopædic Institute of which Professor Putti is Director owes its existence to the generosity and foresight of Rizzoli, a general surgeon of Bologna, who left his whole fortune for the purpose of buying a large Olivetian monastery near the city, converting it to its present purpose, and partially

FIG. 98.—View from the Institute.

endowing it. The Institute is situated on a high hill, about three-quarters of a mile outside the city walls, and commands from its grounds and balconies a magnificent panorama of the city itself and the country for miles around (*Fig.* 98). To English readers the idea of a monastery conjures up visions of frowning walls and dark, depressing cells, but this fine old Italian monastery

is very different; the whole place is full of light and air and beauty, and must serve as a constant inspiration to all who are privileged to work there. Much of the building is of polished marble, and the interior is lavishly adorned with frescoes and mural paintings, whilst its architecture, though simple, is conceived on a noble and effective plan.

The building has lent itself admirably to adaptation as a modern orthopædic hospital. A central corridor runs right through it, and houses on either side the administrative offices and the gymnasia; the wards are disposed to right and left of it, on two floors, arranged about large quadrangles which are planted out as sub-tropical gardens (*Fig.* 99). The adult patients are nursed

FIG. 99.—View in one of the quadrangles.

for the most part in the original monastic cells, which are rooms large enough to accommodate three or four beds; paying patients of the first grade, however, each have rooms to themselves. Children are housed in two large modern wards of the usual type, made by knocking down the parting-walls of a number of the monastic cells.

The hospital can accommodate about 200 in-patients, and these are of two kinds, free patients and contributory patients. The latter are of three grades: (1) Those who can only pay a moderate amount towards the total cost of their maintenance; (2) Those who pay just about as much as they cost; and (3) Those who pay freely. No distinction is made, as regards the general social amenities provided, between the various grades of contributing patients.

The general principle upon which the hospital is run is that their payments, added to the small endowment that was left over from Rizzoli's fortune after the building itself had been bought and adapted, serve to maintain the free patients and to avoid constant appeals to the charitable public. There are, as a rule, about 50 free patients and 150 paying ones; it is found that these proportions are approximately correct in relation to the present economic conditions. Out-patients contribute, being arranged in two grades according to their means, but they do not pay as much as they cost; a few who bring certificates of real poverty are treated entirely gratuitously. The cost of treatment is still high, for the cost of living has not fallen in Italy since the War ended; wages remain at the war-time level, and consequently all commodities remain expensive.

The Director takes an active interest in the business side of his hospital, in addition to carrying out by far the largest share of the surgical work; in his central office he has large-scale graphs, which show all the important administrative data in a form in which they can be readily studied; these are kept hung on the walls all round the room. In this department, also, are preserved the records of all the patients, filed on the card-index system, whilst every sub-department has its own registers and can supply details concerning any patient at a moment's notice. Considerable attention is paid to the keeping of good records, and wherever possible these are made upon a uniform plan; for instance, in all cases of poliomyelitis a special printed form is used on which all the important headings are set down. Full use is made of photography as an aid to accurate recording; almost every patient is photographed before and after treatment, and prints and lantern slides are made from the negatives, and filed according to subject so as to be immediately available for teaching purposes. For the recording of scoliosis Schultess' drawing apparatus is used, in conjunction with a special chart, whilst in many cases of talipes and the like plaster casts are taken and preserved in the museum. The conception and organization of the whole place are admirable, and one recognizes everywhere evidence of the twofold function that the Institute has been designed to fulfil, viz., that of a curative hospital and of a centre for the study and teaching of orthopædic science. And this is surely very fitting, for the city of Bologna is the home of the oldest university in the world. Valsalva and Malpighi were amongst its former professors of anatomy, and the fine old oak-panelled room in which they taught is still shown to interested visitors to the University library, where it serves nowadays as a committee-room; there one may see the beautifully-carved life-size figures of the dissected human body with which the mediæval professors were wont to illustrate their lectures. Here also Tagliacozzi taught, and there is a quaint figure at the head of one of the wooden pillars of the room showing a little imp holding out a human nose towards the visitor!

The University of Bologna is still of considerable size and importance; it has over three thousand students. Dr. Putti is the Professor of Orthopædic Surgery, and gives regular clinical teaching to the undergraduates. At Bologna, Rome, and Naples, medical students are required to attend compulsory courses in orthopædic surgery, though they are not yet required to pass any special examination in that subject.

In connection with the Istituto Rizzoli there is a very fine library, dedicated to King Umberto I. This magnificent room, which was originally the refectory of the monks, has its walls and roof decorated with beautiful mural paintings; its noble proportions and rich colouring make it one of the glories of the ancient city of Bologna. In this library are filed copies of all the leading orthopædic and general surgical periodicals of the world, together with some 7400 theses; and there are over 7800 books of reference upon its shelves. Abstracts are prepared by the librarian, under the Professor's directions, of all the most important papers published, and these are filed and indexed in such a manner as to be readily available. The Director and his associates make considerable use of the library; we were told by one of the younger

FIG. 100.—One of the three gymnasia.

assistants that Professor Putti spent many hours there, studying the writings of foreign authors, or looking up the bibliography of any cases of unusual interest that might be in the wards. His lectures show a wide acquaintance with the work of others, as well as a wealth of personal observation and experience. The fullest possible use is made of all the clinical material available, and so efficient is the system of fact-recording, so fruitful the spirit of progress and research that infects all the workers, that it has recently been found possible to bring out a journal, six times a year, embodying the original papers that emanate from the Istituto. In this journal, entitled *La Chirurgia degli Organi di Movimento*, are also published abstracts of the leading papers in current orthopædic literature.

The basement of the building houses a well-equipped laboratory, in which all routine investigations are made and researches are carried out. There are ample facilities for chemical, histological, bacteriological, and experimental work; at the present moment one of the assistants is engaged upon an investigation of the macroscopical and microscopical changes that take place in transplanted fascia lata and the joints themselves after the performance of arthroplasty in dogs.

The gymnasia (*Fig.* 100) are three in number; there is an ordinary massage and exercise-room, and there are also two instrument-halls, one filled with Zander appliances, and the other with Schultess instruments. Questioned as to his views concerning the efficiency of these merely mechanical methods of exercising, Professor Putti replied that whilst he thought them inferior to the remedial exercises that could be given by a good gymnast, they were at any rate useful in a place where large numbers of patients had to be treated and the staff was limited in number. For example, on the afternoon of our visit 120 patients were expected, whilst during the War there were at one time as many as 800 under treatment.

The museum contains a number of preparations of the ordinary kind, and, in addition, a large collection of plaster casts and small-scale models, specially made for teaching purposes. The casts illustrate every kind of deformity, before and after treatment; amputation stumps; and especially stumps that have been subjected to Professor Putti's operation of cinematization. There is a large exhibit, also, of the artificial limbs that have been devised for use with the cineplastic and other amputations. The models for the most part illustrate conservative methods of fracture-treatment, and amongst the most interesting are the models of Codivilla's original appliances for the treatment of fracture of the femur by direct traction upon the bone. It is claimed by the Bologna school that Codivilla (who was the second director of the institute) was the first man to invent and apply this method.

Professor Putti (*Fig.* 101) is the third Director; he has held the office for about ten years, and served for about fourteen years under his predecessor, Codivilla. He is a whole-time paid officer, and is not allowed to operate or to treat patients outside the Institute, though he may hold consultations outside, and may, of course, have private patients admitted to the institute under his care. He has seven assistant medical officers, the majority of whom are engaged upon some sort of orthopædic research in addition to the routine duties that they perform. The senior assistants perform a certain number of the operations, and, in the case of paying patients, receive a proportion of their contributions for every operation that they perform. They also act as anæsthetists. All plasters, moveover, are applied by the assistants, and they carry on practically the whole of the work in the out-patient department. Professor Putti, it will be realized, is a very busy man, and a man of many parts; he finds time, however, to make himself conversant with the details of the work that is going on in every department, and in every department one can see the impress of his enthusiasm, his vigour, and his progressive, orderly mind. Senior surgeon, administrator, editor, consultant, author, university professor, and head of one of the largest orthopædic workshops in the world, Professor Putti nevertheless finds time to make friends with practically

every patient in his hospital, and to carry out himself the greater number of the operations necessary; and in walking round his hospital with him, it was a pleasure to see the respect and goodwill with which he was greeted by all the patients and their friends.

In connection with the Institute there is, as we have just remarked, a very large workshop, where over 100 men and women are engaged upon the manufacture of artificial limbs, orthopædic appliances of every kind, and even surgical instruments. The patients pay just enough for their appliances to make the workshops self-supporting, without any profit being made. The majority of the artificial limbs required by the Government for army pensioners are made here; there are a certain number of limbs of standard type; and others, of special design, have been devised for individual cases of cinematization. At this factory, also, are produced all the instruments that are used in operation at the Institute, including not only simple tools such as scalpels

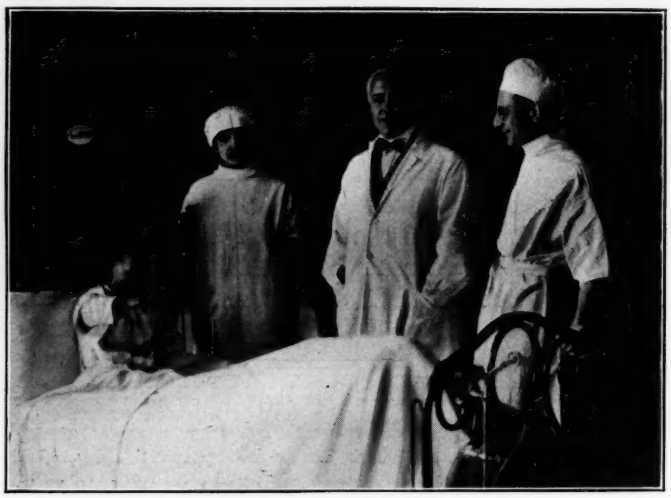


FIG. 101.—Professor Putti in the wards.

and forceps, but also elaborate mechanisms such as motor saws and drills, operating tables, and the like.

The latest development of the hospital is its country branch; a large building has been acquired in the Dolomite Alps for the treatment of surgical tuberculosis and other conditions by altitude and heliotherapy. This building is now in process of adaptation to its new purpose, and will be ready, it is hoped, by August, when 100 patients will be drafted thither from the parent hospital. Motor ambulances will be used, and also special railway ambulance carriages, there being a railhead situated quite close to the new hospital.

Professor Putti is directing his mind more particularly at the present time to the problems of arthroplasty, an operation of which he already has considerable experience. He has performed over 130 arthroplasties, and considers that the knee and the elbow are the two most favourable sites for the operation, with the hip as a good third. Cineplastic amputations he rarely performs nowadays, under peace conditions, and we did not have

an opportunity of seeing this operation carried out; though we saw a patient undergoing re-education, with a temporary limb, after having had the operation done.

We were fortunate in being able to see an arthroplasty of the knee performed, and to study the results of the operation in several patients. The theatre in which the professor works (*Fig.* 102) does not differ in any important detail from any other; it is a large and well-equipped place, all lined with white marble, and furnished with many tiers of seats for onlookers, and ample top-lighting. The flasks of sterile saline solution are kept at body-heat by means of electric hot-plates. Rubber gloves are used, of course, and over these the surgeon and his immediate assistants wear cotton ones; these are changed in the course of an operation as soon as they become soiled, and are frequently moistened with alcohol. Ether is used entirely for anæsthesia, and an admirable feature is the recording, throughout the whole operation, of the

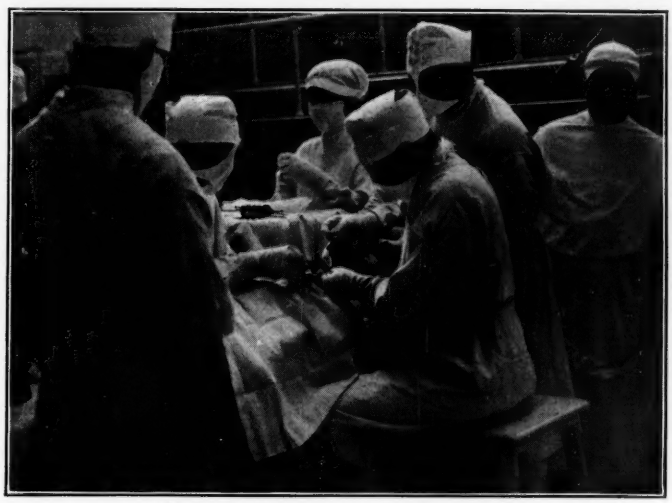


FIG. 102.—In the operating theatre.

brachial blood-pressure; an ordinary sphygmomanometer is used, coupled to a Tycos gauge, but this is of a large size, having a six-inch dial, and is mounted on a tall brass rod, well above the anæsthetist's table, where all can see it readily. It is thus possible to know at any moment precisely how the patient is standing the operation, and the effects of various manœuvres can be watched, and technique improved accordingly. The pressures recorded are charted, together with notes of the stages of the operation with which they correspond, the duration of the anæsthesia, and the total amount of ether used. In a typical arthroplasty of the knee (the first case recorded below), the pressure varied from 100 to 96 mm. of mercury during all the first part of the operation, fell to 80 mm. during the chiselling and rasping of the bones, rose to 98 mm. again as soon this was completed, and remained at 98–100 mm. right up to the conclusion of the operation. The duration of the anæsthesia was one hour, and during this time 320 c.c. of ether were used, given by the open drop method.

The chief assistant at operations is the theatre sister, a very able and highly-trained nurse, who knows every stage of every operation, and has everything required in absolute readiness; the surgeon puts out his hand, and, let us say, a suture of the right-sized catgut, threaded in just the right-sized needle, is at once handed to him. Each assistant and nurse appears to have his own specific duties allotted to him, and there is very little talking in the course of an operation.

OPERATIONS AND CASES.

Case 1.—*Operation.* **Ankylosis of the knee.**

A married woman, age 34, had bony ankylosis of the knee, probably the result of an old gonorrhœal arthritis. A tourniquet was applied, and a curved incision, convex upwards, was made above the knee, prolonged upwards by a straight median incision. Flaps of skin and superficial fascia were turned back. Linear incisions, slightly diverging below, were then made on either side of the patella and the quadriceps muscle, right down to the femur; the tissue between the two cuts was divided parallel with the femur, the superficial portion, attached to the patella, being turned down, and the deeper part turned up. These flaps were wrapped in about twenty thicknesses of gauze, which was kept moist by being squirted from time to time with saline solution from a large ear-syringe.

The joint capsule was divided freely in front of the joint and on both sides, right back to the ligament of Winslow; the latter, however, was left intact, so that, as all subsequent manipulations were carried out inside the joint cavity, there was never any anxiety as to the safety of the main vessels and nerves. Care was taken, however, though the capsule was very freely incised, not to shred it in any way, so that it might heal well afterwards, and the Professor remarked that he never had any fear in any arthroplasty as to the stability of a joint afterwards, but only as to its range of movement.

The crucial ligaments and as much as possible of the soft parts remaining between the femur and the head of the tibia were next cut away. The ankylosis was then divided with special gouges, which were made with cranked handles and a blade that was curved so as to accommodate itself in both dimensions to that of the femoral condyles. The knee was gradually flexed more and more as the division of the bony ankylosis proceeded, the back of the joint being supported on an ingenious little metal crutch attached to the table and raised up by a screw as was required. This padded metal crutch, shaped like an inverted L, we saw utilized also in other operations, in the same manner as a Lorenz wedge, and it was much neater and more convenient than the sandbag that is commonly used to hold a knee in the flexed position when operating. The interior of the 'joint' was now completely cleared, and all remnants of soft coverings and articular cartilage were removed. The surfaces were finally smoothed with rasps and files, and shaped as nearly as possible like normal condyles.

The head of the tibia was now similarly treated, special care being taken to preserve a good high ridge, running antero-posteriorly, between the two tuberosities; the hollows on either side of it for the femoral condyles were made, if anything, rather deeper than normal. By the time that both bones were fully shaped, there was a space of about one-third of an inch between them.

A long straight external incision was next made on the outer aspect of the thigh, and a flap of deep fascia was dissected up and entirely detached. This flap was transferred to the knee, and fitted perfectly, without any trimming; it was placed with its superficial aspect towards the interior of the new joint cavity, and covered the whole of the condylar surface of the femur and the head of the tibia. It was pressed well to the back, so that its reflexion lay in contact with the posterior ligament of the joint, and was then secured in place by a number of single suture-points of fine catgut, attaching it to the capsular ligament of the joint.

The under surface of the patella was then denuded of cartilage, filed, and covered with a flap of soft parts reflected from the tongue of tissue originally left attached to it. Two slits, sagittally placed, were then made in the fascial graft: one over the tibial spine, and the other opposite to it, over the corresponding intercondylar notch of the femur. This was for the purpose of allowing connective tissue to form between the two bones at this point, and the surgeon stated that he knew for a fact that it did so form; in two cases in which he had had cause to re-open a joint on which he had operated, he had actually seen the dense white fibrous tissue which united the bones; though not crucial, these fibres nevertheless formed efficient interosseous ligaments.

The quadriceps muscle was now resutured with lock-stitches of catgut, and the skin with interrupted salmon-gut sutures. Dressings were applied, the tourniquet was removed, and the limb placed on a plaster back-splint, flexed to 30°, which had been previously prepared. As soon as the patient was put back to bed, a heavy weight-extension was applied, the knee being kept flexed to the same extent as before.

Case 2.—**Arthroplasty of knee 4½ years previously.**

The patient had undergone the operation of arthroplasty of the knee 4½ years previously for ankylosis following a gunshot wound of the joint. He was a captain of infantry, about 30 years of age. As he walked to and fro, it was almost impossible to detect which was the injured leg; he was able to stand quite steadily on the operated leg, and said that he could walk for 'many kilometres'. He is still in the army. His range of movement is 180° to 80°. There is slight grating in the joint, but no pathological mobility; fluid is absent. There is still considerable wasting of the quadriceps (over 1 in.), and the Professor remarked that seldom, if ever, was the full bulk of this muscle restored.

Case 3.—**Arthroplasty of knee 2½ years previously.**

A man, age 27, had undergone arthroplasty of the knee 2½ years previously for a severe post-typhoid ankylosis. There had been originally a severe *B. typhosus* pyæmia, and not only had the knee-joint actually suppurated, but there had been osteomyelitis in the femur as well, and a number of peripheral abscesses. The femoral condyles had undergone a certain amount of destruction, and in this case the result was not so favourable as in the first; there was some lateral play, and knock-knee, which the patient thought was tending to increase.

The Professor frankly admitted that the result in *Case* 3 was not nearly as good as in *Case* 2, though pointing out that conditions here were comparatively unfavourable. He explained that he classified his results as 'good', 'fair', or 'bad', according as they had movement from 180° to 90°, from 90° to 45°, or less than this, and claimed that up to the present he had produced 54 per cent of 'good' results in the case of arthroplasty of the knee.

The joints of both these patients yielded, on palpation, a coarse grating identical with that which one experiences on examining a typical osteo-arthritic knee. Fluid, and thickening of the soft parts, suggestive of fringes, were totally absent, however. The radiograms showed, as the Professor pointed out, changes that were extremely like those of osteo-arthritis: some condensation of the articular surfaces where pressure was sustained, and proliferation at the free margins; in one case, actual osteophytes were present, one of which seemed to be loose; and on two occasions, we were told, it had been necessary to re-open the joints to remove osteophytic loose bodies. It was pointed out that some of the plaques of bone seen near the free margins of the femur and tibia were not really free, as they appeared to be, but were situated in the attachments of the capsule to the bones; Professor Putti drew an

analogy with the traumatic ossifications that are sometimes seen in the bony insertions of muscles, which, as he said, are not true periosteal ossifications (for true periosteum is not present where muscle tendons are inserted), but fibro-osseous ossifications.

Case 3 illustrated a further point of practical importance, namely, the great value of retaining a good median ridge on the tibia, and good concavo-convex modelling of the articular surfaces. Where these are secured all tendency to lateral luxation of the tibia is absent, whereas if they are absent some such play may be present. No difficulty has ever been experienced through defective re-formation of the articular capsule and ligaments; they always form perfectly. As a matter of fact, however, the capsule is only very slightly damaged in the operation as described above; it is only cleanly divided in the horizontal plane, and re-unites easily.

Case 4.—**Arthroplasty of the knee 10 years previously.**

A man, age 40, had had arthroplasty of the knee performed ten years previously for ankylosis following pyo-arthrosis secondary to osteomyelitis of the femur which had perforated into the joint. In spite of the severity of the initial infection, a very good functional result had followed upon the operation. He walked without a limp, and he told us that he could cycle thirty-five miles and back, every week, to fetch his week's pay. After the War, he served for two years in the army in a trench-filling party! At the present time he was engaged as a farm labourer. His knee was chronically swollen, and his quadriceps somewhat wasted; some grating was present, moreover, on movement of the joint. But abnormal mobility was completely absent, and the joint moved freely within the range 180° to 90°.

Case 5.—*Operation.* **Congenital talipes equinovarus.**

This was a relapsed case of ordinary congenital talipes equinovarus in a girl, age about 11. The foot had been fully redressed and plastered three weeks previously. It was now proposed to transplant the tendon of the tibialis anticus to the outer side of the foot to prevent the relapse of deformity that would otherwise have been inevitable.

The tendon was detached at its insertion, and tied very tightly at its extremity with a ligature of fine wire; the object of this was to prevent its fraying out after the subsequent fixation. It was pulled up above the annular ligament, and then passed down to the outer aspect of the foot by means of an ingenious tunnelling instrument. This consisted in a hollow tube of metal, slightly flattened, through which was passed a loop of tough wire; by slipping this loop over the end of the tendon, and then dragging it down tightly against the smooth end of the tube, the tendon was held firmly, and yet left undamaged. A small incision over the fifth metatarsal exposed the bone, as far forward as possible; the periosteum was raised over a small area, and the tendon was then secured to the bone by driving through it a fine screw, $\frac{5}{8}$ in. long, having a small washer underneath it. The limb was then put up in plaster by an assistant in the over-corrected position.

The Professor said that he preferred this to any other method of tendon fixation; he always fixed tendons to bone, never to one another.

Case 6.—*Operation.* **Talipes following old sciatic paralysis.**

The tibialis posticus was passed behind the tibia to the outer side, and affixed there with screw and washer, as above. The tendons of the flexor longus digitorum were then tenotomized, the toes well manipulated, and the limb plastered.

Case 7.—*Operation.* **Double congenital dislocation of the hips in a girl, age 10.**

There had been no previous treatment. The radiogram showed a well-formed acetabulum and neck of femur, but a head that was lying very considerably above

the proper place. The child, deeply anæsthetized with ether, was placed on a high block of the form shown in *Fig.* 103, the hip resting over the excavation, and Professor Putti proceeded to manipulate it into place, stretching the resisting muscles, but neither hacking nor dividing them. In carrying out this manipulation, he rested his hand upon the child's knee, and his chin upon his hand, explaining that he found it easier in this way to estimate just how much force he might safely employ. He emphasized the importance of fairly rapid reduction, dwelling upon the profound shock that was caused by protracted wrenching. In this particular case the manipulations did not occupy more than three minutes, and the spread-eagle plaster was rapidly applied by an expert assistant; nevertheless, when we saw the child next day, she was still showing evidence of fairly severe shock, though she recovered completely within the following twenty-four hours.

FIG. 103.

Other cases of general interest, which we saw in the wards, were: one of tuberculosis of the lumbar articular processes in a young man; several cases of congenital dislocation of the hip in various stages of conservative treatment; a very early case of coxa plana; and three examples, from three separate families, of hæmophilic knee, which had formed the subject on the previous day of the Professor's clinical lecture to the undergraduates.

In the laboratory, one of the assistants was kind enough to demonstrate to us the knee-joint of a dog on which he had performed arthroplasty a month previously. The transplant of deep fascia was firmly adherent everywhere to the underlying bone; its surface was slightly roughened, like a piece of 'matt' surface drawing-paper, but was not villous, nor actually ulcerated. We saw, also, some microscopical sections of this transplant: the original structure of the fascia was very little altered, but in some of the interfibrillary spaces, just deep to the articular surface, there was a well-marked leucocytic infiltration. We were told that in two cases of arthroplasty of the knee in which it had been necessary to re-open the joint, the transplant presented a very similar appearance to that which we saw in this experimental preparation. The operation of arthroplasty has been performed upon a series of dogs, and it is proposed to make a complete study of the gross and microscopical changes that take place at various dates after the performance of the operation.

FEMORAL HERNIA, AND THE SACCULAR THEORY.

By R. HAMILTON RUSSELL, Melbourne, Australia.

The operation for femoral hernia should be a very simple procedure. I wish to deprecate most earnestly what would seem to be a growing tendency to operate for femoral hernia from above Poupart's ligament; I am convinced that this is taking a great hazard, without any compensating advantage whatever. Accidents will most assuredly happen, accidents involving the femoral vein and others involving the bladder; moreover, it is, I believe, most important to avoid interference with the fascial structures surrounding the upper opening of the crural canal. Anything we do there will inevitably

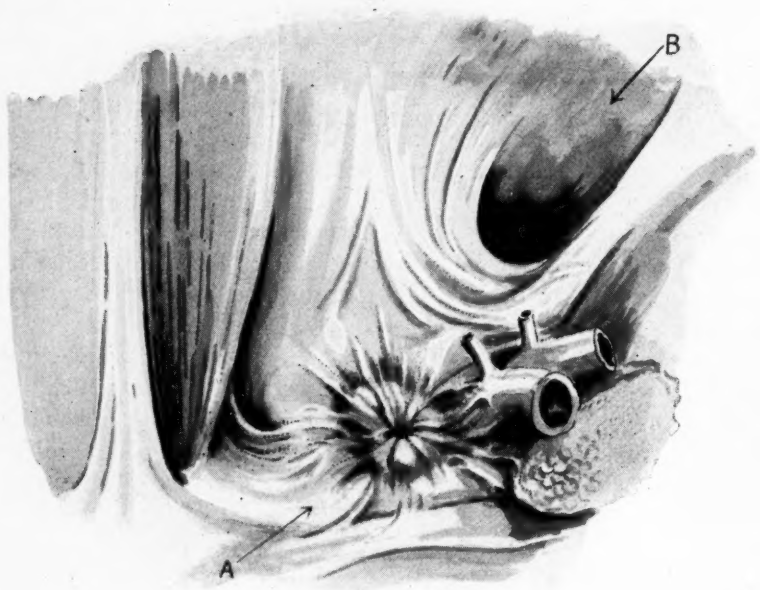


Fig. 104.—Internal view after closure of femoral sac by torsion.
A, Femoral sac closed. B, Inguinal canal.

make the opening larger, never smaller; and there is no protecting muscular sphincter like that which guards the inguinal canal.

Femoral hernia occurs as the result of the presence of a congenital sac in the crural canal; it occurs under no other conditions, and it will be cured by the removal—or, a better word still, the *abolition*—of the sac. The sac has only to be abolished without doing any damage to the upper opening of the canal in order to abolish the hernia; but it is quite possible, through a lack of wise circumspection, so to enlarge the upper end of the canal through

our manipulations that a fresh hernia might actually descend into it, which, strictly speaking, would merit the name of a traumatic hernia.

I venture to urge a trial of the following simple procedure, which I have employed for many years.

Operation.—The sac having been exposed in Scarpa's triangle in the usual way, is first cleanly defined up to the margin of the saphenous opening. It is now opened, and should there be a strip of adherent omentum it must be released and pushed back into the abdomen. Next the sac is seized with a pressure forceps and firmly torsioned; the twisting will complete the separation of the sac from the margin of the saphenous opening, so that the twist will extend up the canal and close the mouth of the sac at the femoral ring. The twisted sac may now be crushed and ligated with catgut at a convenient spot, but no determined attempt should be made to pull the sac down as far as it will come, nor should the aim of the operator be directed necessarily to reaching the uppermost limit of the sac; his object will be to apply the ligature a little lower down, for the mouth of the sac is not to be closed by the ligature but by the twist. So much of the sac as is redundant is then cut away, but the remainder of the twisted sac is pushed up the canal pretty firmly and left there, aiming to substitute a convexity for a depression at the site of the crural ring (*Fig.* 104). The operation is completed by closing the saphenous opening with one or two catgut stitches, and finally applying a firm compressive dressing. I myself always use over all a rubber bandage (Martin's bandage) very gently applied (barely on the stretch).

Theoretical Considerations.—The theory provides for:—

1. Closure of the mouth of the sac at the femoral ring with the minimum of interference with the fascial structures there.

2. Complete abolition of the sac.

3. *Safety.* The greatest danger to be apprehended in operating for femoral hernia has been shown by experience to be injury to the bladder. This accident has happened many times, and sometimes surgeons have been courageous and public-spirited enough to record such misadventures. It appears to me that the plan of torsioning the sac should render any such accident to the bladder practically impossible, seeing that by no means could any portion of the bladder enter the twist or escape notice.

The Practical Application of Surgical Principles in Oblique Inguinal and Femoral Hernia contrasted.—This little study will be found both curious and instructive. The two herniæ have in common the fact that they are both dependent upon the presence of a congenital sac in the inguinal and femoral canals respectively; but they are singularly divergent in almost every other feature. The inguinal sac is, from its mouth downwards, in close relation with the vas deferens and vessels of the cord; so that in order to detach these structures it must be handled with the greatest freedom. It must be pulled forcibly out from under cover of the abdominal muscles while the detaching finger separates the cord structures deeply into the pelvis. The sac is firmly torsioned, crushed, and ligatured *at its upper extremity.* The fascia transversalis receives scant consideration, and protection from recurrence is provided by the musculature.

Now contrast the indications for dealing with the femoral sac. There are no cord structures and there is no musculature. The fascial upper boundaries of the crural canal must be held sacred; there must therefore be no forcible pulling down of the sac, or they will be stretched and damaged. Hence the sac cannot be ligatured at its neck, like the inguinal sac; the mouth of the femoral sac is closed by torsion from below, and then the twisted sac is pushed into the canal, and kept there by any simple device the surgeon may choose; I myself prefer gentle elastic pressure outside the dressing.

HISTORICAL NOTE ON FEMORAL HERNIA AND THE SACCULAR THEORY.

It will be noted that in the foregoing article I have taken for granted the dependence of femoral hernia upon a preformed congenital sac. There ought, I submit with all deference, to have been no need for this note. I have been, however, amazed to find on consulting some of the most recent text-books that the fact of the congenital origin of femoral hernia still seems to be shadowed by uncertainty and doubt in the mind of some authors.

That this is due to the opposition directed against the saccular theory of hernia by so eminent an authority as Sir Arthur Keith I have no manner of doubt; and it would be unreasonable to suppose that an opinion so weighty should be without substantial influence upon the views of anatomists and surgeons. But there is another side to the question. There are some facts of anatomy, and even of embryology, that come more readily into the visual field of the surgeon than into that of the anatomist and the embryologist. It was Sir Berkeley Moynihan who gave us our first lesson in this line of thought some years ago, with his brilliant little essay on *The Pathology of the Living.*

In the matter under discussion the following are historical facts; but I particularly wish to emphasize that I have no thought of animadverting upon our anatomical brethren.

1. For centuries anatomists had the field of femoral hernia to themselves: a field that was not contested by the operating surgeon, who only operated for the relief of strangulation, and never for femoral hernia *per se*; and anatomists completely failed to recognize that the sac was a congenital structure and the essential cause of hernia.

2. Towards the end of the last century surgeons first began to operate for the cure of femoral hernia, and within a decade or two the fact that the sac was a congenital structure was noted and demonstrated by myself, an operating surgeon, and communicated to the Australasian Medical Congress of 1902.[1]

3. An incidental remark made in that communication to the effect that hernial sacs would be found in the sites of hernia if a sufficient number of bodies were examined, arrested the attention of R. W. Murray. He took me at my word, examined 100 bodies, found the sacs as predicted in surprising numbers, and thenceforward became a powerful and convinced champion of the view that the cause of all the ordinary spontaneous varieties of hernia is a congenital sac at the site of the hernia.[4]

4. During the years immediately following, thanks to the interest displayed by Professor (now Sir Harry) Allen and Professor Berry—his successor

in the Chair of Anatomy at the Melbourne University—in addition to femoral sacs, obturator sacs were discovered on several occasions in the dissecting room. In this way there was also discovered a curious little direct inguinal sac that may be found with moderate frequency coming through the conjoined tendon. It was previously quite well known that a direct hernia would sometimes come through the conjoined tendon; but it was not known that the cause of its doing so was the presence of a congenital sac in that situation. How it comes there I know not, but it is an anatomical fact, and was described and shown in the Presidential address to the Medical Society of Victoria in 1903.[2]

5. In 1906 "The Saccular Theory of Hernia" was published in the *Lancet*,[3] and was energetically criticized by Sir Arthur Keith. A discussion carried on at 'a distance of weeks' between England and Australia was obviously impossible, and it was agreed to leave the question to the test of time and experience, with the words, "The saccular theory will fail or prevail as it is false or true".

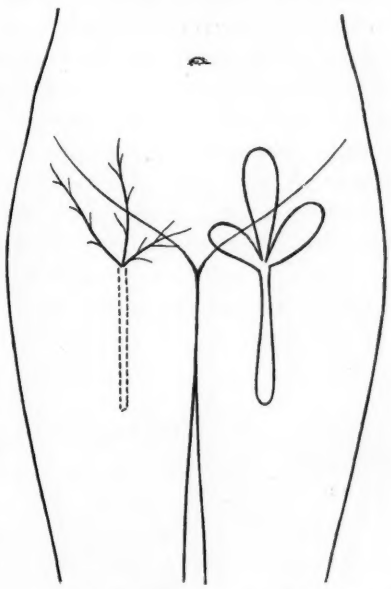


FIG. 105.—*Right side*, branches of common femoral artery. *Left*, positions assumed by femoral sacs.

6. A somewhat long jump from 1906 to 1923. During these years I believe I may safely say that the saccular theory 'prevailed' and became firmly established among surgeons and surgical writers; but I felt less confident as to the views held by teachers of anatomy. All the greater therefore is the significance I attach to an article in the *Journal of Anatomy* for January, 1923, where there appears the report of an investigation into the subject of femoral hernia by J. Allison Panton.[5] Dr. Panton undertook this inquiry with the object of testing the truth of the prevalent theory as to the reason for the greater frequency of femoral hernia in the female. After a laborious study, carried out with an industry and patience beyond all praise, he amassed a volume of evidence bearing on the accepted theory; but he also did something more, and better. It is a little difficult to make out how much or how little he had the saccular theory in mind at the beginning, but it is evident that he was amazed at the end of his work to find that he had clearly demonstrated the congenital origin of the femoral sac.

I have no desire to prolong this article by attempting to record the evidence already advanced in proof of the congenital origin of the femoral sac, but I should like in conclusion to re-state one matter of surpassing interest which is concerned with the course taken by a femoral hernia of any size after its emergence from the saphenous opening. No explanation worthy of the term had ever, prior to 1902, been advanced by any surgeon or

anatomist as to the reason why a femoral hernia should usually turn upwards over Poupart's ligament, but should sometimes pass in the opposite direction down the thigh, sometimes outwards toward the iliac spine, and sometimes inwards toward the pubes. It is disappointing to read in a modern text-book the explanation that "the hernia follows the path of least resistance", or words to that effect. How can there be four different "paths of least resistance"? The explanation becomes at once simple and obvious as soon as we recognize that the sac is a congenital structure. The accompanying diagram (*Fig.* 105) has already been used by me on two occasions.[2, 3] It shows on one side the branches of the common femoral artery, and on the other the various positions assumed by femoral herniæ when large enough; and the size of a femoral hernia is determined by the size of the preformed sac far more rigidly than is the case with inguinal hernia. Now it takes very little thought to determine how the arterial branches depicted have come by their curious retrograde course; when once that is understood all difficulties with regard to the behaviour of femoral herniæ vanish. A sac contemporaneous with the developing arteries in the embryological limb-bud will be subject to the same developmental evolutions and will be compelled to take up the same positions; so far as one can see, it would appear to be a matter of mere chance into which of the four positions the sac will be drawn. So the 'path of least resistance' may be a perfectly correct expression to use in describing the course of femoral hernia, but solely on one condition, which is that the word 'hernia' shall be taken as meaning the hernial contents only, and shall not include the sac; for the 'path of least resistance' is the interior of the sac.

REFERENCES.

[1] Russell, R. Hamilton, *Lancet*, 1902, May 31.
[2] Russell, R. Hamilton, *Ibid.*, 1904, March 12.
[3] Russell, R. Hamilton, *Ibid.*, 1906, Nov. 3.
[4] Murray, R. W., *Ibid.*, March 10, 363.
[5] Panton, J. Allison, *Jour. of Anat.*, 1923, Jan.

MALIGNANT DISEASE OF THE UPPER JAW: WITH SPECIAL REFERENCE TO OPERATIVE TECHNIQUE.

By E. MUSGRAVE WOODMAN, Birmingham.

(Being the Hunterian Lecture delivered at the Royal College of Surgeons of England on January 31, 1923.)

In the year 1671, Richard Wiseman, Sargent Surgeon to King Charles II, describes an operation on the upper jaw in the following words :—

" A man about 28 years of age came out of the country with a cancer on his left cheek stretching itself from the side of his nose close under the lower eyelid to the external canthus, so making a compass downwards. It was broad in its basis, and rose, capped like a sugar loaf. The cancer threatened his eye with inflammation, and he hastened up and importuned me to undertake it. I complied with his desire, and four or five days after, having prepared all things ready, the actual cauteries, digestives. desensitives, and bandages, I pulled the tumour towards me with one hand and made my incision close to the eyelid and cut it smooth off. Then viewing our work and observing some relic of cancer remaining above the external canthus, we consumed it by actual cautery and dressed the wound with our digestives, with embrocation, desensitives, and moderate bandages ".

This is the earliest extant account of an operation in the upper jaw which I can trace, and in a simple way foreshadows the improved technique as it is known to-day. Undertaken as it was centuries before the introduction of anæsthetics or before the value of antiseptics was conceived, the operation demanded a courage and patience on the part of the sufferer which is amazing.

At a far earlier date, Ambroise Paré gives a comprehensive and accurate account of the clinical appearance of malignant disease in the upper jaw, accompanied by the remark that it must not be touched by the hand because it savours of the nature of a cancer. Percivall Pott displayed a very considerable interest in polypi of the nose, and gives an incisive description of the difference between simple and malignant cases, but directions for operative treatment are conspicuous by their absence. In the works of Hunter and Abernethy I can find no record of work done in this sphere. To Professor Lizzars, of Edinburgh, is attributed by Liston the credit of introducing the formal operation of removal of the upper jaw.

Liston himself undertook numerous operations of this description, and the following maxim dominated his work :—

" The more rapidly, consistent with safety, these operations—which under any circumstances are of great severity and attended with much suffering to the patient—be accomplished, the better."

The late Sir Henry Butlin took a great interest in this difficult field, and did much to increase the success of operative treatment of malignant disease in the jaws. Himself a great operator, his work on the anatomy and surgery of the lymphatic glands of the neck, especially in reference to malignant disease, has proved of great value to those who come after him.

FIG. 106.—Section of an endothelioma arising from the ethmoid, showing the rather advanced vacuolation in the cells.

The confines of space necessitate a ruthless restriction of the field that can be covered in this paper. Pathology can only be lightly dealt with, and the clinical side of the subject can hardly be touched at all on the present occasion.

Classification.—I have attempted to classify the malignant neoplasms in the upper jaw according to the site of origin, as follows :—

a. Palate and alveolus.

b. Air sinuses.

c. Epipharynx, with invasion of the jaw.

d. Cheek, with invasion of the maxilla.

FIG. 107. — Typical spheroidal-celled carcinoma. Notice the cells arranged in large acini, with a fibrous stroma starting each group.

Pathology.—In no part of the body are such varied and extensive types of growth to be found, and almost every histological structure enters into their composition. The lower portion of the superior maxilla takes part in the formation of the mouth, and consists of dense bone covered by fibrous periosteum and a stratified squamous epithelium. In these tissues sarcomata of all varieties are common and rapidly malignant,

but fairly accessible to treatment. On the other hand, an epithelioma is moderately slow in growth, ulcerates, and spreads quietly until it perforates through the palate or alveolus into the antrum. It may be taken as a general rule that any growth arising around the antrum is drawn as it were to a centre, and sooner or later seeks this inviting space.

Of the many interesting and rare tumours of the teeth and dental papillæ, I can add nothing to the work of Sir Anthony Bowlby, Sir John Bland-Sutton, or Sir Frederick Eve.

The second function of the upper jaw is entirely different, and it has to do with respiration and the resonance of sound. To fit it for these duties the bones are delicate, vascular, and covered by a ciliated epithelium. The tumours arising in this portion of the maxilla do not often contain bone. Macroscopically they are usually soft, succulent, friable, and vascular, and there is little difference between the morbid appearance of sarcoma and carcinoma. It is very difficult to obtain any reliable figures as to the point of origin of the growth. I believe in the majority of cases the tumour originates in the ethmoid, and only invades the antrum as a line of least resistance.

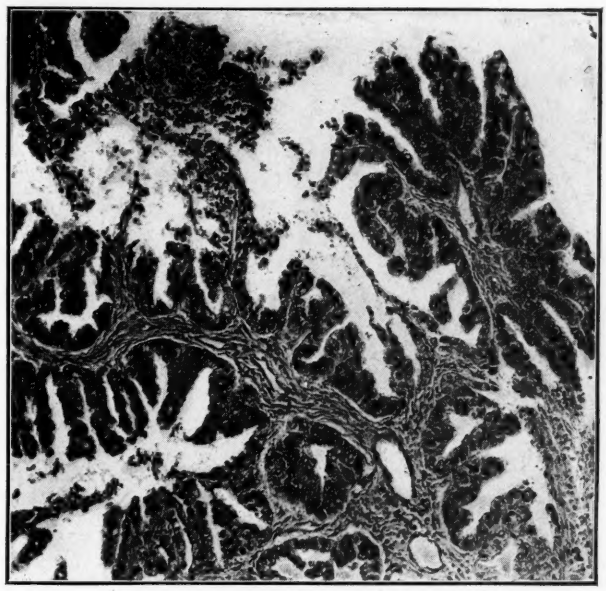


FIG. 108.—A columnar-celled carcinoma, showing typical branching processes covered with columnar cells. The general appearance almost suggests a bladder growth.

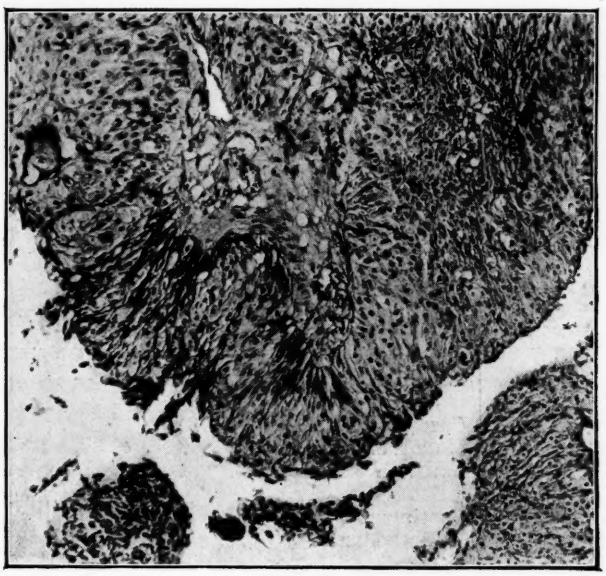


FIG. 109.—A squamous-celled carcinoma of an unusual type. The section through various portions of the growth showed in one place a very aberrant type of squamous-celled carcinoma, in another an ordinary epithelial papilloma, and in yet another a fibromatous condition in which the projection was covered by a single layer of squamous epithelium.

Consider the anatomy of the ethmoid for a moment. Composed of a mass of small cell-spaces

with septic infection retained in little pools throughout its substance, the conditions are eminently favourable for the development of malignancy. The antrum, on the other hand, is a clean sinus usually full of air and lined with ciliated columnar epithelium, endowed with the property of sweeping away and to a large extent of destroying infection.

FIG. 110.—A somewhat atypical section of an epithelioma arising in the alveolus, which had filled the antrum. In the present section the cells have largely become vacuolated and undergone degeneration, but in other parts of the growth there were typical cell nests. The section illustrates the difficulty in microscopic diagnosis.

E. D. D. Davis states that in no single case, out of a series of twenty, could it be definitely shown that the disease arose in the mucoperiosteum of the antrum. If this proposition is correct the significance is obvious; the ethmoid lies outside the superior maxilla, and mere removal of this bone will not eradicate the growth.

Turning to microscopic anatomy (*Figs.* 106–110), there is some divergence of views as to the relative frequency of the various types (*see Table I*), and this is due to the histological difficulties encountered.

Table I.—TYPES OF GROWTH.

NAME	NO. OF CASES	TYPE OF GROWTH	
Mollison ..	17	Carcinoma ..	14
		Sarcoma	3
Harmer ..	23	Carcinoma	18
		Endothelioma ..	5
Davies ..	21	Carcinoma	10
		Endothelioma ..	7
		Sarcoma	4
Woodman ..	30	Carcinoma	8
		Endothelioma ..	6
		Epithelioma ..	8
		Sarcoma	8

Many tumours are atavistic; in others an entirely different appearance is present when sections are taken from separate portions of the same growth.

Complications.—True to its instinct, a growth when it has reached the limits of the antrum erodes the bony wall where resistance is lowest. It not infrequently extends upwards through the floor of the orbit. A proptosis of the orbit results, and is not serious unless accompanied by injection of the vessels and œdema of the conjunctiva, when invasion of the sphenoidal fissure is indicated.

Case 1.—M., male, age 47. This patient came under my care with a recurrent malignant growth in the right upper jaw. The growth had extended upwards, and could be felt beneath the lower eyelid. It projected from the orifice of the nostril in the form of a bleeding and somewhat fungating mass.

The superior maxilla was removed, including the orbital plate, and the growth did not extend into the pterygomaxillary fossa, but upward into the ethmoid, and backward into the sphenoid and frontal sinus, which contained pus and polypi, and a small bud of growth was just beginning in the lumen of the cavity (*Figs.* 111, 112). The operation area was seared with the diathermy button.

On examining the naked-eye specimen the growth was found to have perforated the infra-orbital margin and to project into the cavity. Microscopically it proved to be a malignant endothelioma eroding the bone (*Figs.* 113, 114).

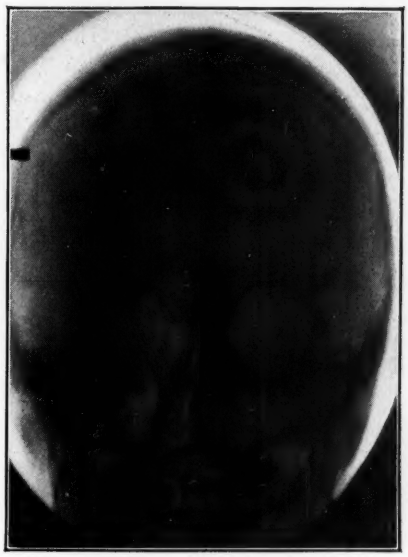


FIG. 111. — *Case* 1. X-ray photo, antero-posterior of skull, showing a completely dull right antrum, ethmoid, frontal sinus, and the whole of the side of the nostril in shadow.

FIG. 112.—*Case* 1. X-ray of the skull, showing the shadow in the frontal sinus and the sphenoid sinus.

Partial or complete blindness may be due to toxic neuritis of the ophthalmic nerve, and is not a contra-indication to operation unless accompanied by atrophy. A further point at which the bone of the antrum is thin is situated just below the infra-orbital margin, and here invasion into the soft tissues of the cheek is common. On the other hand, the bony floor of the antrum is dense, and invasion of the palate from above is rare, and growth only appears on the alveolar margin through the socket of a tooth.

If we assume that malignant disease usually commences in the ethmoid, the invasion of the other sinuses is readily explained. The sphenoid is closely connected with the posterior ethmoidal cells and is often continuous with them, while the duct

leading to the frontal sinus is in direct relation with the anterior portion of the ethmoid. Whether the frontal sinus is often the seat of growth is a matter of dispute; but in some cases it certainly is so, and in most the frontal sinus is the seat of suppuration. The sphenoid is involved in the majority of malignant cases by direct extension.

FIG. 113.—*Case* 1. Microscopical section of the growth, showing the typical vacuolated cells of an endothelioma.

Involvement of the meninges is a serious and often hopeless complication. When it is remembered that the roof of the ethmoid is formed by the thin plate of bone forming the floor of the anterior cranial fossa, it must be evident that in the complete eradication of disease the dura will be exposed on many occasions and often over a considerable area. Needless to say, no operation should be undertaken in the presence of meningeal infection; but it is important to determine whether this infection is due to sepsis or to growth. Sepsis usually accompanies malignant disease of the jaw, and all the uninvaded sinuses become full of pus.

Case 2.—Mrs. B., age 50, was sent to me from the country with double ethmoidal polypi. Examination of her nose showed it to be completely full of rather vascular fleshy polypi on both sides, and all the sinuses were dull. She complained of headache and general ill health. I operated in the upright position for polypi, and found them very extensive in distribution and highly vascular. On microscopical examination they proved to be myxosarcoma, and the basal portions were filled with numerous sarcomatous cells. They recurred very rapidly, and ten days afterwards I reflected the cheek on both sides and exenterated all the sinuses, every one of which on both sides of the nose was found to be full of growth. She suffered from a severe attack of meningitis which nearly terminated her life; but she recovered, and has now remained free from growth and is in perfect health. Operation July, 1920.

FIG. 114.—*Case* 1. Another portion of the growth, showing erosion of the orbital margin.

Lastly, the accidental wounding of the dura mater of the anterior fossa is not of necessity a dangerous proceeding.

Case 3.—A., male, age 37. This patient had an extensive epithelioma of the cheek involving the eyelids and invading the ethmoid and antrum (*Figs.* 115, 117). The whole area, including orbit and upper and lower lids, was excised (*Fig.* 116). When removing the growth from the dura mater in the region of the cribriform, a $\frac{1}{2}$-in. incision was accidentally made in the dura with scissors, and cerebrospinal

FIG. 115.—*Case* 3. Extensive ulcerated area involving the whole of the right orbit. Note the indurated masses of the growth.

FIG. 116.—*Case* 3. The patient after operation, showing the whole area of the orbit exenterated, and the wound area covered by skin.

FIG. 117.—*Case* 3. Colour microphotograph of the growth, showing columns of epithelial cells radiating in different directions, with a marked cellular reaction of the invaded tissue.

fluid escaped. The edges of the dura were sterilized with iodine and carefully sutured, and the wound was packed with a drainage tube leading down to the dura mater. Uninterrupted recovery followed, without even a rise of temperature or a headache.

FIG. 118.—*Case* 5. Photograph, full face. Note the broadening of the base of the nose, with the deviation of the axes of the eyes outwards, and the pitting of the skin where it was adherent to the growth over the bridge of the nose.

The last and perhaps most difficult extension to deal with is backwards into the pterygoid fossa. Here the dense muscles of mastication are involved, and the growth worms itself into the loose vascular and areolar tissue of this region and leads early to deep lymphatic involvement.

Case 4.—T., male, age 42. An ulcer was excised from the angle of the cheek on the left side near the upper wisdom tooth. This proved to be epitheliomatous, and a year later he developed pain in the face, and a sinus leading from the antrum freely discharged pus in the region of the previous operation. An X-ray examination showed the left antrum and ethmoid to be dull. The upper jaw was excised on the left side, and the growth was seen to have perforated into, but only partly filled, the antrum. It had, however, extended widely backwards into the region of the external pterygoid muscle beneath the malar bone and entered the sheath of the temporal muscle. The latter extensions were destroyed by diathermy. The patient made a good recovery, and is at present free from recurrence.

Radium.—The use of radium is of comparatively recent date, and the results are still *sub judice*. Radium attacks the more highly specialized cells less than the primitive ones; a round-celled sarcoma is far more vulnerable than any other type of tumour, and when attacked by a dose of emanation it often melts away like an inflammatory mass.

Case 5.—S., male, age 42. This man had a slowly but actively progressing sarcoma growing from the base of the skull, which had eroded the ethmoid on both sides, destroyed the septum, and was pushing out the nose and tilting the orbits outwards so that conjugate vision was impossible (*Figs.* 118, 119). The growth was a giant-celled sarcoma (*Fig.* 120). It has apparently been entirely destroyed by radium, and the nose is contracting slightly. The man is well and in regular work.

FIG. 119.—*Case* 5. Photograph, side view, showing the obliteration of the sulcus at the bridge of the nose.

Case 6.—F., age 27. Patient was admitted to hospital with a large swelling on the left side of the palate and over the supratonsillar fossa. This appeared to be a gumma, and resembled a quinsy, but there was no pain. Swelling extended so

rapidly that a tracheotomy was required in the night, and a section was taken. This proved to be a small round-celled sarcoma growing from the posterior ethmoidal region and blocking the back of the nose. In the mass 100 mgrm. of radium were buried. The whole tumour was destroyed, the tracheotomy tube removed, and the patient discharged (*Fig.* 121). Three weeks later he came back with an exactly similar condition in the right side of the soft palate. Radium was placed in this swelling, and it also disappeared within twenty-four hours. The patient was again discharged, and came up a month later with a large fixed mass in the supraclavicular fossa. Radium being buried in this, the tumour once more disappeared. The prognosis is bad, it being almost certain that the growth will appear in the mediastinum and probably prove fatal.

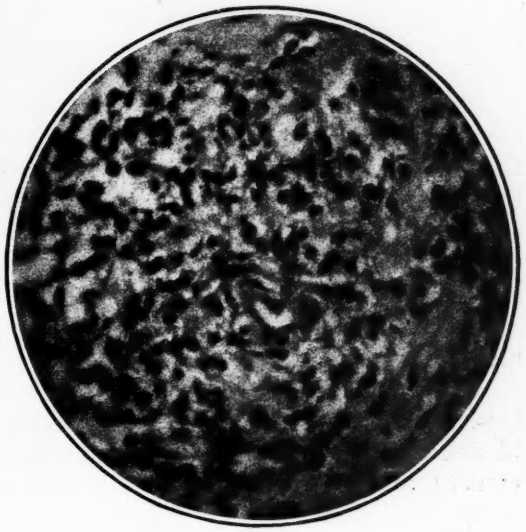


FIG. 120.—*Case* 5. Microphotograph of the growth. It is a giant-celled sarcoma, with a typical multinuclear giant cell in the centre.

Radium has a distinct value in dealing with sarcoma, but in only a few cases will it cure.

The tumour should be exposed and radium buried in the substance, while a flat applicator, properly filtered, is placed externally so as to give a cross-fire of radiations. Preference is now largely given to small glass tubes of emanation introduced throughout the growth and placed in the form of a barrage around the edge. Radium has little value in dealing with a carcinoma or epithelioma.

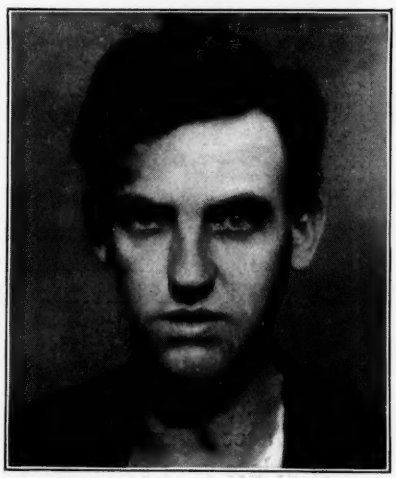


FIG. 121.—*Case* 6. The patient after treatment, showing the healed tracheotomy scar.

The question of possible damage to the eye when radium is placed in the ethmoid region is a matter of some importance. Fortunately radium has little effect on the highly specialized cells of normal tissue, and damage to the sight is extremely rare.

My experience of the new Erlangen method of X rays has not been so happy as that of radium. Finzi states that the whole question depends upon giving every cell of the growth a lethal dose, and this dose varies according to the kind of growth. The lethal dose for round-celled sarcoma is less than that for a spindle- or mixed-celled sarcoma. The question of sepsis complicates the situation, and most growths are septic. It does no harm to give an aseptic growth considerably more than the lethal dose, but there is considerable harm in giving more than a lethal dose to a septic growth, and failure

results. When the growth is infected it should be sterilized with ionization, and treated on the following day by intensive X rays.

Diathermy.—The use of diathermy marks a real surgical advance in the treatment of malignant disease in certain regions. The aim is to sterilize the whole growth by a process of coagulation necrosis.

In a cancer of the breast or bowel it is possible to excise the disease completely by passing through healthy tissue—and here diathermy has no field. On the other hand, in disease of the mouth or throat, it is often impossible to avoid contamination of the wound by cancer cells, which are sown on to the raw surface, and it is to the effusion of infected blood and lymph that this is largely due. The advantages attending the use of diathermy are many. There is an absence of implantation of malignant cells. The cut surfaces of the blood and lymph vessels are sealed. There is no loss of blood, and shock is very slight.

The proceeding is simple. The growth is transfixed with a pronged electrode, and the current turned on up to $1\frac{1}{2}$ ampères. The tissue becomes white, blanched, and dry. The current is then switched off before sparking commences, and a further area is attacked, and the coagulated tissue is broken away with forceps. Finally the whole surface of the excavated area is sterilized with the button electrode.

There are certain disadvantages which must be considered :—

1. Owing to coagulation proceeding beyond the area of the electrode, vessels and nerves cannot be seen.

2. During the application of the current ether must not be used as the anæsthetic.

3. Although at the end of operation the affected area is sterile, it does not remain so, and there is a period of sloughing which, in the case of tendon tissue or of bone, takes a long time and is often very offensive during the process of separation.

4. Secondary hæmorrhage may occur. The rule should always be to ligature the vessel when diathermy is used in the neighbourhood of a large artery.

In order to overcome these disadvantages the best results can be obtained in the upper jaw by a combination of diathermy with surgery. The growth should be fully exposed, destroyed by diathermy, and then removed, and the raw surfaces of bone and soft tissue sterilized by the electric current.

OPERATIVE TECHNIQUE.

In dealing with the operative technique, I desire at the outset to call attention to the necessity of modifying the operation according to the site and extent of the growth. In an epithelioma of the palate it is sufficient to remove the half of the palate and alveolus involved, unless the antrum has been perforated and invaded by growth. In an alveolar sarcoma a similar conservative operation will be sufficient, and no advantage whatever would accrue from removal of the infra-orbital plate. On the other hand, a growth in the antrum probably arises from the ethmoid, and the operation of removal of the superior maxilla is totally inadequate.

The air-cells form one indivisible unit; from the bottom of the antrum to the roof of the frontal sinus, and from the pterygomaxillary fossa to the posterior wall of the sphenoidal sinus there is a continuous chain of epithelial cells; thus the ramifications of this system extend far beyond the confines of the superior maxilla, and sufficient elasticity must be given to the operation to allow for alteration in design to meet each individual case.

Fig. 122.—*Case* 7. G., age 35. A very extensive endothelioma of the right superior maxilla, which had ulcerated through into the mouth and upwards into the cheek over the maxilla. The growth was traversed with numerous fistulous tracts and impregnated with pus. Operation was impossible and radium refused.

The difficulty of successfully removing a growth in the upper jaw lies in the fact that two of the three cardinal principles of removal of malignant disease are usually impossible to carry out. It is generally impracticable to remove the growth through healthy tissue, and, secondly, it is not possible entirely to prevent dissemination of diseased blood over the wound area; but it is just here that the assistance of diathermy is most valuable.

There are certain classes of cases which are generally inoperable (*see Fig.* 122):—

1. Sarcoma arising from the base of the skull and secondarily involving the maxilla.
2. Extensive involvement of the pterygomaxillary fossa.
3. Cases showing persistent meningeal infection.
4. Extensive invasion of the back of the eye suggesting involvement of the cavernous sinus—in which case the removal of the eye will not cure the disease.

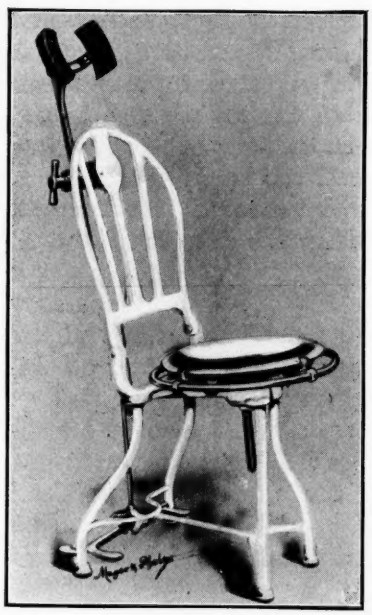


Fig. 123.—Chair used in upright position, which can be instantly lowered at will by the pressure of a foot pedal.

The usual practice is to put the patient when anæsthetized on the chair in the horizontal position and carefully raise it to the vertical by drawing forward the back of the chair.

On the other hand, involvement of the skin of the face or of the eye itself is not necessarily a contra-indication.

Position.—There are two matters at this point which deserve some attention. In the first place the posture of the patient during operation is important. The upright position has a great deal to recommend it. First of all, the blood-pressure to the head is considerably reduced, and there is an absence of nasal congestion. Secondly, the visibility is greatly improved, and not only can the operator see exactly what he is doing, but the steps of the operation can be clearly seen by those around. In the third place, there is a remarkable absence of shock, the patient often leaving the table

with a good pulse. And lastly, the amount of anæsthetic required to keep the patient at a sufficient depth is very much less than in the recumbent position. With a properly equipped chair (*Fig.* 123) the position presents no difficulties.

Anæsthetic.—The anæsthetic used is a matter of very material importance to the operator, and the latest advances in the use of intratracheal ether (*Figs.* 124, 126) have, in my opinion, reduced the mortality to as low as almost any operation in surgery.

The technique now made use of is shortly as follows: Induction takes place by ethyl chloride and ether, and when the patient is fully anæsthetized the mouth is opened and the post-nasal plugs are carefully packed into the back of the nares, and a large intratracheal catheter is passed through the larynx under vision. An anæsthetic airway the proper length and curve is then inserted at the back of the pharynx, and the mouth packed with gauze. Lastly, a sterile towel is passed across the mouth and tacked over the airway and passed round the back of the head. Ether vapour is then pumped in from the tube in the mouth directly to the lungs under a pressure of 20 mm. of mercury by an electric motor, and the anæsthetic is maintained indefinitely by positive pressure. There are two distinct advantages for this class of operation in the use of intratracheal ether. There is no danger whatever of the inspiration of blood, a minimum quantity of ether is used, and an even depth of anæsthetic is maintained. During the last ten minutes oxygen is blown in, and at the completion of the operation the swallowing reflex should always be present.

FIG. 124.—*Case* 8. W., age 15. Patient in vertical position under anæsthetic. Note the intratracheal tube coming in from the left side and the airway.

This patient was sent up with a repeated and severe epistaxis. On examination he was found to have an extensive sarcoma growing from the sphenoid, involving the right ethmoid, and extending into the antrum on the corresponding side.

Chloroform is never given unless the use of diathermy necessitates its employment.

Operation.—The incision commences above in the centre of the eyebrow, and it is carried downwards midway between the bridge of the nose and the inner canthus of the eye, and thence follows the line of Ferguson's incision down the groove at the side of the nose and round the external naris to reach the philtrum, and thence divides the lip. On the buccal surface of the cheek the greatest care should be taken to divide the mucosa low down immediately above the neck of the teeth and to elevate it throughout the

whole length of the incision. Thereby a considerable portion of harmless mucous membrane is saved, and can be sutured in position to the raw area on the inner side of the reflected cheek. The cheek flap is then drawn aside, and care is taken to carry the knife down through the periosteum to the bone, particularly on the inner side of the nose. If the growth has extended backwards, and especially if its base is in the pterygoid muscles, it is necessary to make use of the horizontal incision beneath the orbit, but this should be avoided where possible. The cheek flap should be protected from infection during the process of removal of the growth, and the raw surface is swabbed with tincture of benzoin and protected with a small gauze pad soaked in the solution and sewn into position. The subsequent steps depend upon the nature, origin, and extent of the growth. The operation must be carefully planned in its exact details before the patient comes to the theatre, and there should be no delay or hesitation in the technical details at this stage.

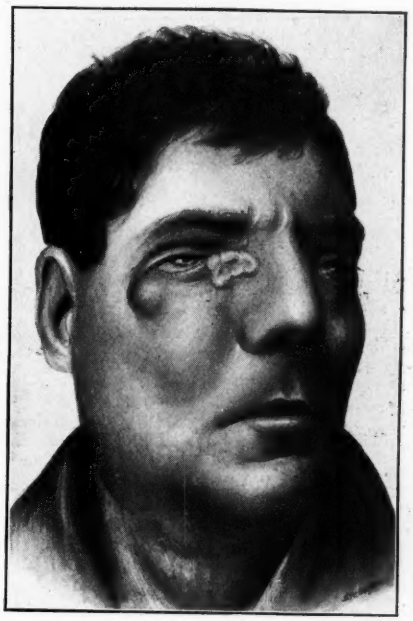


FIG. 125.—*Case* 9. B., male, age 46. An extensive endothelioma had arisen from the ethmoid; the growth had perforated through the superior maxilla and appeared under the skin, which it had infiltrated to the outer side of the eye, and the sight was lost on this side. In this case the upper lip was saved and detached from the cheek, the skin being united to the mucous membrane throughout the primary incision. The growth was then isolated above from the forehead, round the malar bone, and the whole superior maxilla together with the orbit, the ethmoid, and the nasal bones removed *en masse*. Extensions were then seen to have taken place in the frontal sinus and in the sphenoid, These were removed, and the wound was closed over as far as possible. A period of time must elapse before any further operative treatment can take place. I then propose to close the wound in the face by plastic flaps to reconstitute the orbit.

In this figure is seen the patient before operation. Note the nodules of the growth beginning below the outer canthus of the eye, at the side of the nose, on the upper eyelid, and at the inner end of the eyebrow. The steps of the operation are shown in *Figs.* 126–130.

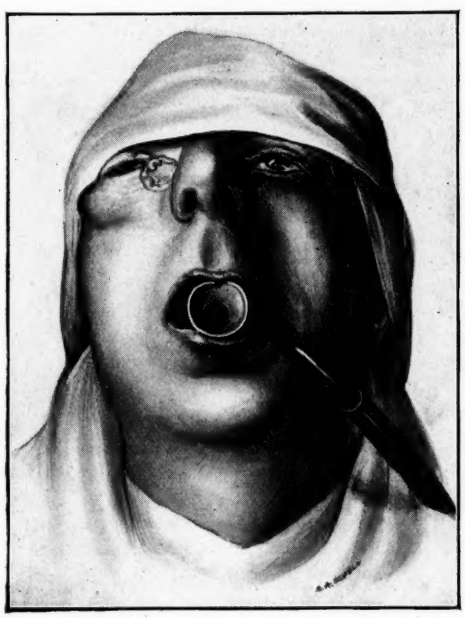


FIG. 126.—*Case* 9. Patient under anæsthetic in the vertical position, anæsthetized by intratracheal ether.

If the growth is confined to the lower half of the superior maxilla and does not involve the upper air sinuses, the lower part of the upper jaw is removed, leaving the infra-orbital plate intact. To do this a fine chisel is taken and a horizontal incision made following and parallel to the lower margin of the orbit at about the level of the infra-orbital foramen. This is carried through the ascending nasal process of the superior maxilla to enter the nose, and through the body of the malar bone to the pterygo-maxillary fossa. The line of attachment of the cartilages of the nose to the bone is then divided, and if the nose itself is not involved the muco-periosteum can easily be elevated and the whole of the soft parts of the nose turned inwards without opening the cavity. The hard palate is then divided sagittally from the alveolar process backwards. The separation is completed by detaching the soft palate by a horizontal incision, and lastly separating the back of the maxilla from the pterygoid process by driving a strong osteotome in between these bones. This partial incision, when it is adequate, gives very satisfactory anatomical results; the orbital cavity is not opened, and there is no dropping of the eye with consequent failure of alinement. Further, the nasal cavity is also unopened and its important functions remain intact. This proceeding is suitable for most growths arising from the palate and alveolus, even when they have perforated the antrum, provided the limitations of the growth can be accurately seen and delineated.

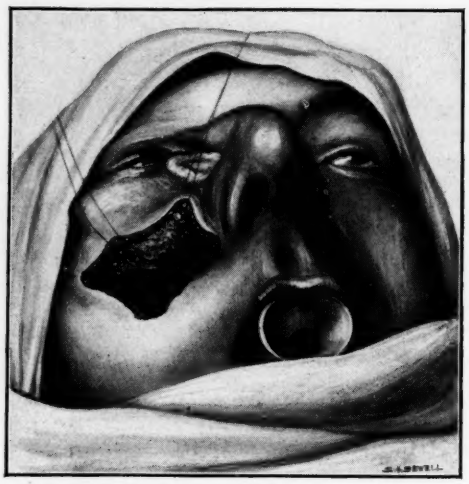


FIG. 127.—*Case* 9. Incision of the soft parts below the growth, and suture of the skin below the mucous membrane of the mouth, so as to preserve the upper lip.

FIG. 128.—*Case* 9. The incision round the growth extending down the side wall of the nose and above the eyebrow and across the malar bone. The soft tissues have been incised down to the bone in all directions.

Very different is the proceeding which must be adopted when the malignant changes have involved the upper air sinuses or the orbit, in which case a most extensive exposure is required to eliminate the disease. The complete upper jaw must be removed in the first place, and the whole of the side wall of the nose. Only too often this proceeding is considered sufficient for removal of the growth,

but it cannot be too strongly emphasized that this is a stage in the access to and exposure of the deeper and more delicate parts around the skull base.

By no operation is it possible to remove such a growth in one piece without breaking across various extensions, and a very brief consideration of the anatomy would easily convince the surgeon that this is so. The whole of the ethmoid up to the cribriform plate should be systematically removed. Then the sphenoid should be opened, the anterior and inferior walls of the sinus cleared away, and the contents exenterated. In a similar manner the frontal sinus must be dealt with. The duct is traced upwards, and all the fronto-ethmoidal cells and the whole of the floor of the frontal sinus are removed. It is never advisable or necessary to remove the anterior wall; thus considerable deformity is saved and, what is even more important, the risk of infecting the diploic veins avoided.

FIG. 129.—*Case* 9. Superior maxilla excised together with the ethmoid and a little of the orbit. The dura mater of the anterior fossa was exposed and is seen in the upper part of the wound area. The fat of the pterygomaxillary fossa is shown in the lower portion.

FIG. 130.—*Case* 9. Wound narrowed somewhat by bringing in the flaps after the slough has come away. The large granulating area of the wound was completely covered by a skin graft.

Several cases of osteomyelitis have been recorded as the result of Killian's method of exposure of this sinus. If possible, an endeavour should be made to leave the periosteum of the orbital cavity intact, but nothing must be sacrificed to the complete eradication of the growth. Special attention must be paid to the fat and muscles of the pterygomaxillary fossa, and to a common extension of the growth backwards through the internal naris to enter the pharynx, where it lies free in the cavity. Extension into the pterygoid fossa I regard as being the most difficult to remove, and one of the most frequent causes of recurrence. Having perforated the thin posterior wall of the antrum, the growth finds itself in a region highly vascular, and it spreads rapidly between the fasciculi and planes of the pterygoid muscle into a region where access is bad and elimination very difficult. After healing takes place, there

is often a residual fibrosis in these muscles which leads to considerable difficulty in opening the mouth.

At the conclusion of this complete operation, the frontal sinus, the sphenoid, and cribriform plate should lie freely exposed and form one large cavity leading to the mouth below, limited internally by the septum of the nose and externally by the replaced cheek flap.

In the course of the operation little trouble will be experienced from hæmorrhage. The vessels on the cheek flap are easily caught as they are divided. On the removal of the superior maxilla, which should be carefully done between the fingers, the internal maxillary artery is brought forward in the loose fat, and can often be clamped with curved tonsil forceps before being divided. If divided it can very easily be picked up and ligatured.

If there is contamination of any area in removal of the growth, the best course is to run over the whole part with a flat button diathermy electrode.

Lastly, in cases of sarcoma it is always wise to insert radium in the operation area for twenty-four hours after operation.

In suturing, great care must be exercised in the region of the inner canthus of the eye. The periosteum should be picked up and sutured as a separate layer, and the skin gradually approximated by a continuous stitch. The dressing which has proved most effective is one of gold leaf. A clean healing wound results, and no other dressing is required after the first twenty-four hours.

Method of Dealing with Lymphatic Glands.—It is a matter for discussion whether it is wise or necessary to remove the glands in all cases. It is a fact that in many growths of the superior maxilla no glands are affected—especially is this so of the carcinomata. On the other hand, with a squamous epithelioma of the palate or alveolus the glands should always be completely dissected out, following the technique so ably laid down by the late Sir Henry Butlin. It is not possible to be sure which group of glands will be affected, or, in fact, on which side of the neck they will appear.

Case 10.—S., age 20. Primary endothelioma of the septum. The only gland involved was the superficial parotid on the opposite side of the neck, and the microscopic appearance was that of the primary growth.

The microscopic diagnosis of the tumour will be of some assistance in deciding the point in each individual case, but in the absence of palpable glands the operator will probably prefer—and rightly—to await the signs of glandular infection, though naturally a close watch must be kept over the patient.

Ligature of the External Carotid Artery.—Another point of debatable interest is the question of the desirability of ligature of the carotid artery before operation. Sir Henry Butlin found that ligature of the external carotid was of no use alone, and Sir F. Treves holds that preliminary ligature of a large artery is not a necessary or desirable proceeding. In my own case, for the last two years I have not tied the artery.

Laryngotomy.—Until quite recent years laryngotomy was an indispensable preliminary to attacking the upper jaw. The airway was then free

and untrammelled by the actions of the surgeon, the anæsthetist was out of the way, and it enabled the pharynx to be plugged off with sponges so as to preclude the entrance of blood to the air-passages. But is there *no* disadvantage in making a direct opening into the air-tubes? Additional operating time is involved, there is a cricothyroid artery which sometimes is troublesome, and there is always a remote possibility of blood leaking past the sponges in the throat. It seems certain, therefore, that in the future we shall make use more freely than ever of intratracheal ether, and it is to this anæsthetic, and to this alone, I attribute the remarkably low mortality of the modern operation.

FIG. 131.—*Case* 11. W., male, age 26. In this patient a soft, vascular, round-celled sarcoma grew from the ethmoid, involving the antrum and the frontal and sphenoid sinuses. The whole facial wound suppurated afterwards, and the hole is seen opposite the inner canthus of the eye; and deformity is seen below, due to removal of the orbital plate and subsequent dropping of the eye. This opening will have to be closed by a plastic operation.

After-treatment.—The main principles to be held in view are to obviate deformity and to prevent recurrence. Dealing with the question of deformity first—experience teaches that there are certain situations where trouble may be expected. In the first place, at the inner angle of the eye the skin of the side of the nose is thin, delicate, and easily curls inwards and lies in contact with the periosteum of the nasal bones, and it is only too easy to leave an unsightly hole in this position (*Fig.* 131).

FIG. 132.—*Case* 12. C., male, age 26. This patient had a primary epithelioma of the palate, which had perforated into the antrum and filled this cavity with growth. The upper jaw was removed, together with the palate. The orbital plate was left intact. Note the absence of facial deformity resulting.

To prevent this the skin incision should be made well on to the side wall of the nose and away from the eye, and the greatest care taken in the elevation and retention of the periosteum, which should be subsequently sutured back in a separate layer to the skin.

Œdema of the eyelid is a great and constant bugbear, and is sometimes unavoidable. By omitting the horizontal and infra-orbital portion of Ferguson's incision I hoped to avoid this deformity, but without marked success. The determining factor appears to be the orbital plate of the superior maxilla (*Fig.* 132). Where this can be left, œdema of the lid is not permanent; but where it has been removed, however careful the operator is not to disturb the ligament of Lockwood or the attachments of the capsule of Tenon, the eye as a whole drops and the under surface acquires fibrous attachments to the tissues forming the posterior wall of the wound area. To obviate this it is recommended that the temporal muscle be split vertically down to and through the coronoid process of the lower jaw, that this musculo-bony tissue be swung across beneath the orbit like a sling, and the

bone be fixed to the nasal septum or to the nasal process of the frontal bone. I have carried this out in a recent case with success.

In some cases it is quite possible to save the palate, when the comfort of the patient and the rapidity of his recovery are correspondingly increased. In other cases a good result can be obtained by reflecting the mucoperiosteum of the palate inwards to the middle line and suturing this to the cheek flap after removal of the bone.

FIG. 133.—Plaster case taken from the mouth before operation. In the first place the mould was taken in wax, and the plate made from this to represent the plaster case of the mouth itself.

But the advantages of a foramen in the palate cannot be overlooked—it enables the whole operation area to be open to inspection, it facilitates free drainage, and allows efficient dressing after operation. I am convinced that in the majority of cases it is both advisable and necessary to leave this opening; for a view is obtained stretching from the frontal sinus to the back of the sphenoidal cavity and from the pterygomaxillary fossa to the pharynx. A recurrence can be readily detected and exterminated with precision.

In regard to dental restoration a few words will suffice. A few days before operation a plaster cast must be taken of the patient's mouth by a dental surgeon interested in this work (*Fig.* 133). A temporary denture can be fitted within a few hours of operation (*Fig.* 134), greatly facilitating the feeding of the patient and mitigating his discomfort. Such a splint should have a bulbous extension upwards roughly to fill up the cavity of the antrum and keep the cheek out in its normal position. A final denture is fitted after a few months.

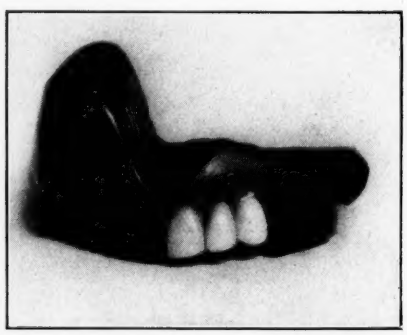


FIG. 134.—Temporary dental splint. This is made of vulcanite, with a bulbous extension reaching into the cavity resulting from removal of the jaw. This can be inserted within forty-eight hours of operation, and enables the patient to take his food.

Space does not permit me to go into the details of plastic restoration. If adhesions have already taken place below the eye, they should be freed, and a stent epithelial mould inserted and the cheek flap kept up by a denture.

The disfiguring hole at the inner angle of the orbit can be remedied by swinging up a portion of the upper part of the septum, and fixing it in position; in a short time this should be covered with epithelium, or a plastic skin-flap be turned down from the scalp. More extensive plastic efforts are required when the skin of the face has been removed, and each case must be planned out according to its special requirements. It is a fundamental point that no plastic operation to cover a disfiguring hole should be undertaken until all reasonable danger of recurrence is past, and the whole cavity should be freely open to inspection for at least a year after operation.

RESULTS.

Mortality.—During late years the mortality of this operation has shown a remarkable decrease. In view of the extensive removal of tissue, of the division of large sensory nerves near their ganglia, of the loss of blood, and of the septic wound which overhangs the air-passages and sometimes involves the food tract, this is extraordinary. It is, I believe, due to the use of intratracheal ether.

Table II.—Mortality Statistics.

Operator	No. of Cases	No. of Deaths	Percentage
Butlin	?	?	33·3
Göttingen Clinic ..	84	17	20
Harmer	23	2	8·5
Mollison	17	1	6
Davies	21	0	0
Woodman	30	0	0

Growths arising primarily in the retropharyngeal space and extending into the nose or superior maxilla from behind come into a different category, and often are best approached from the mouth by reflexion of the palate, though on some occasions they can very easily be removed by primary resection of the superior maxilla. The operative mortality of these growths is considerably higher than that of growths arising in the superior maxilla or air sinuses, and this is probably due to the presence of a septic wound overhanging the air tract: in my own series I have had three deaths from septic pneumonia or sepsis extending down the neck.

Recurrences.—In a fairly large proportion of cases there are recurrences. There are two ways in which malignant tissue can be left *in situ*. The whole of the growth may not have been exposed—such, for instance, as an extension into the frontal sinus—and for this I can offer no excuse. In the second place, some implantation of cancer cells or infected blood is likely to take place over the raw surface of the flaps in the best planned operation, and this is unavoidable.

I feel sure that improving technique will produce just such an improvement in the statistics of recurrence as has already taken place in those of mortality, and that at no distant date.

If the operation be so planned as to leave the whole sinus area open to free inspection, the seriousness of a recurrence is reduced to a minimum. Unlike the recurrence of cancer in any other part of the body, the implantation deposit is less serious and more easily cured than the primary disease.

Table III.—RECURRENCES.

NAME	NO. OF CASES	TYPE OF GROWTH	RECURRENCE	NOT TRACED
Mollison ..	17	Carcinoma .. 14 Sarcoma .. 3	16	— —
Harmer ..	23	Carcinoma .. 18 Endothelioma 5	16 5	— —
Davies ..	21	Carcinoma .. 10 Endothelioma 7 Sarcoma .. 4	6 0 4	— — —
Woodman ..	30	Carcinoma .. 8 Endothelioma 6 Epithelioma .. 8 Sarcoma .. 8	3 1 4 2	1 0 2 2

CONCLUSIONS.

1. The pathology of the neoplasms of the upper jaw is not yet clear, but there is a high degree of local malignancy in nearly all types.

2. The operation to be employed for removal of disease in the upper jaw should be modified to suit the case—it should be based on an intimate knowledge of the anatomy of the air sinuses and a sound surgical technique, together with a close study of the disease in the patient to be dealt with.

3. Intratracheal ether as an anæsthetic has great advantages, and will replace laryngotomy.

4. While mortality has been reduced to a minimum, recurrence is still an ever-present fear. The operation should be so designed as to facilitate inspection and the eradication of recurrent growth, and no attempt at plastic repair should take place until this fear is dispelled.

THE CLOSURE OF THE SUPRAPUBIC URINARY FISTULA FOLLOWING SUPRAPUBIC PROSTATECTOMY: OBSERVATIONS ON 68 CASES.

By H. P. WINSBURY WHITE, London.

The 68 cases which are dealt with in this paper are from a series of 77 consecutive cases operated upon at St. Peter's Hospital between April and November, 1921; of the 9 cases not included, none survived convalescence.

The purpose of this paper is to consider: (*a*) All the factors which have a possible influence on the time of closure of the suprapubic urinary fistula; and (*b*) The utility of the in-dwelling catheter.

The subject is dealt with under the following headings: (1) Brief outline of post-operative treatment; (2) Division of cases into groups in relation to the employment of an in-dwelling catheter; (3) The use of the in-dwelling catheter; (4) The operative procedures; (5) The onset of micturition; (6) The time of removal of the suprapubic drain; (7) Secondary hæmorrhage; (8) Malignant cases.

1. **Brief Outline of Post-operative Treatment.**—The parietal and bladder wound is closed round Freyer's tube[1] at the end of the operation, and in some cases a small drain is inserted into the prevesical space. Freyer's tube is left draining the bladder for three or four days, and then replaced by a smaller one.

Urine is drained into suprapubic cellulose dressings,[1] maintained with a many-tailed bandage; the dressings are changed four-hourly. When the prostatic cavity is packed at operation, the gauze is removed on the third day. Daily irrigation of the bladder and prostatic cavity is carried out by the suprapubic route and by Janet's method.[2] On the tenth day, the suprapubic drain and sutures are removed and a large steel sound is passed per urethram. The patient is sitting up out of bed during the third week in an uncomplicated case. Or an in-dwelling catheter is used when advisable. The patient is discharged from hospital when the fistula has closed.

2. **Division of the Cases into Groups in relation to the Employment of an In-dwelling Catheter.**—The following classification has been found convenient:—

Group I.—All cases whose fistulæ were finally closed by the twenty-eighth day without an in-dwelling catheter.

Group II.—All cases showing signs of delay in closure of fistulæ, subdivided as follows: (*a*) Treated with in-dwelling catheter; (*b*) In-dwelling catheter contra-indicated for the time being. The cases under (*b*) terminated in one of two ways: (i) Spontaneous closure in due course; (ii) Closure following the delayed use of an in-dwelling catheter.

Into *Group I* fell 38 per cent, the average number of days required for closure being twenty; the earliest occurred on the thirteenth day.

The cases under *Group II* (*a*) were treated with an in-dwelling catheter for three days at some period during the fourth week of convalescence. There were 28 cases—about 41 per cent. The result in 19 cases (67 per cent) was complete closure of the fistula before the twenty-eighth day; of the remaining 9 cases, it is interesting to note that 6 had not commenced micturition up to the time the catheter was tied in. The use of the catheter in such cases will be referred to later.

Group II (*b*) refers to those cases which would have benefited by an in-dwelling catheter but which showed some contra-indication for its use. They were 13 in number, or 20 per cent of the total. As already mentioned, these terminated either by closing spontaneously, or not until after the delayed use of the in-dwelling catheter. Of the 13, 8 (about 61 per cent) subsequently closed without an in-dwelling catheter, the average period being thirty-seven days; 5 (about 38 per cent) were delayed until a catheter could be borne with safety, the average number of days before closure being thirty-four. It will be noted that the fistulæ of the cases in which the in-dwelling catheter was used closed sooner than the others, thus demonstrating its value.

The following were the conditions which supervened and prevented or delayed the use of an in-dwelling catheter: (1) Acute epididymitis; (2) Pyelonephritis; (3) Slough or phosphatic deposit on the wound surfaces. By far the commonest of these was acute epididymitis, accounting for 7 of the 13, about 54 per cent. The part played by acute epididymitis in determining delay depended on whether the complication supervened early or late in the convalescence. If the former, there was not necessarily a contra-indication for the catheter by about the fourth week. In a previous paper entitled "Epididymitis and Suprapubic Prostatectomy: a Study of 50 Cases", the writer[3] showed that 64 per cent of acute cases occurred during the first week of convalescence.

In the few cases in which an in-dwelling catheter was tried in the presence of some degree of pyelonephritis, the result was always to increase rather than diminish the signs of infection. It was therefore found more satisfactory in such cases to dispense with the catheter entirely.

With regard to slough or phosphatic deposit on the wound surface, in a few cases a coating of phosphatic deposit occurred. It appeared early, and commenced to peel off as a fine slough about the end of the third week. An in-dwelling catheter could serve no useful purpose until the granulations were free from slough. Moreover, these cases were rather prone to acute epididymitis and pyelonephritis. In other cases it was unusual for the part of the parietal wound closed by suture not to heal by first intention. The open portion generally showed healthy granulations by about the seventh day. Two-stage prostatectomy cases, however, were an exception to healing by first intention, as the wound invariably broke down in the subcutaneous area.

3. **The In-dwelling Catheter.**—The ideal sought and encouraged was undelayed closure without the aid of an in-dwelling catheter; but in many cases where fistulæ persisted without giving any indication of closing, this was established at once by a judicious use of the catheter; and again, in

cases of delay in recommencing micturition, the in-dwelling catheter re-established the habit, which was an essential before closure would occur.

The reason for avoiding the use of the catheter, if possible, is that it is a foreign body in the urethra and granulating prostatic cavity, and in the presence of already existing sepsis tends to increase it.

The urethritis set up appears to be in proportion to the length of time the catheter is retained. No amount of care can prevent a purulent discharge from the urethra which has borne a catheter for six days. The discharge is serous until about the third day. By prolonging unduly the use of an in-dwelling catheter in the hope of providing an efficient dependent drainage, it should be borne in mind that the value of the procedure is being minimized to some extent by the inevitable sepsis which results from the presence of the catheter in the urethra. In this series of cases the practice was followed of removing the catheter at the end of the third day: in none of these cases did any complication arise. On the other hand, it is not difficult to produce acute epididymitis by leaving a catheter in position for a week. Moreover, the vesical end of the catheter becomes coated with urinary salts.[4] In order to get the maximum benefit in the minimum of time, it is essential that the catheter be not resorted to too soon.[5] When any doubt arose as to whether a case was ready for an in-dwelling catheter, it was generally advisable to postpone its use for a day or two. Employing the catheter too soon means leaving it in position longer than intended. A urethritis is thus set up which might have been avoided. Any question of the fistula becoming epithelialized in the meantime was safeguarded by curetting it with a sharp spoon, while drawing together the edges of the wound with adhesive plaster was helpful in bringing about closure in many cases.

Before final closure of the fistula can be accomplished by the use of an in-dwelling catheter, it is necessary that the wound surface be free from slough or phosphatic deposit, and that micturition be re-established.

These factors being present, then the most favourable moment is when the fistula remains dry for an hour or more at a time. In such cases closure from an in-dwelling catheter can be relied on. On the other hand, it may be said that once a case has reached the 'wet and dry' stage, spontaneous and final closure is imminent and the catheter is not required. This was certainly so in some cases; but in several, where an in-dwelling catheter was contra-indicated for other reasons, the fistula remained open for a considerable time subsequently, in spite of these indications. The plan followed, therefore, was to resort to the catheter if spontaneous closure had not occurred after several days of this stage, provided there was no contra-indication for so doing.

Where the spontaneous onset of micturition was delayed, it was essential to resort to the in-dwelling catheter. In some this was done when micturition had not commenced by the twenty-first day, but in no case did final closure of the fistula result after removing the catheter until it had been used a second time following a week's interval of rest for the urethra.

Fistulæ most obstinate in closing even after the use of an in-dwelling catheter were noted in the following cases: (1) After the second stage of prostatectomy when preliminary cystostomy had been performed several

months previously, and when a cicatrized fistula remained; (2) Where re-establishment of micturition did not occur until after the use of the in-dwelling catheter.

Gum-elastic coudé, ranging from 18 to 22 French scale, were the catheters employed. The largest size was always used where possible, as it provided the best drainage. It was seldom that one so small as a No. 18 had to be employed, and such cases always required a good deal of attention, as the narrow lumen easily became blocked. When this occurred, the only satisfactory way to deal with it was to remove the catheter and replace it by another. Obviously, all the good intended from the catheter may be lost if it is allowed to remain blocked for several hours, as the increasing intravesical pressure may cause the fistula to re-open. It was essential for the success of this part of the treatment to make frequent inspections and irrigations.

4. **The Operative Procedures.**—These embraced three types of operation: (1) Freyer's operation; (2) Thomson-Walker's operation; (3) Two-stage prostatectomy.

One or other of the first two methods was employed in 81 per cent of the cases, and on an average the fistulæ in these cases closed on the twenty-sixth day. In the remaining 19 per cent, prostatectomy was performed in two stages, the fistulæ closing on an average on the thirtieth day; but the slower cases were by no means all in the last group.

With the more tardy ones the question naturally arose whether the delay was due to any obstruction to the outflow from the urethra. In several of these, on passing a metal sound per urethram, an obstruction could be made out between the prostatic cavity and the bladder.[6] In all of these cases Freyer's operation had been performed. As it did not fall to my lot to pass the sound in all cases, I cannot give the actual figures.

In two-thirds of the total number of cases operated upon, Thomson-Walker's operation[6] was performed, which entirely eliminated the possibility of obstruction.

In the two-stage cases the intervals between the cystostomy and the removal of the prostate covered periods varying from two weeks to eight months. Two cases had prostatectomy as long as eight months after cystostomy, and, as would be expected, in each the fistulous track was considerably fibrosed at the time of operation. One took eight weeks and the other nine weeks to close.

5. **The Onset of Micturition.**—Apart from several exceptional cases, micturition did not recommence until several days after the suprapubic drain had been removed. The practice followed was to remove it on the tenth day unless there was an indication for continuing the drainage a little longer.

As stated by Sir John Thomson-Walker[7]: "Occasionally there is a rise of temperature when the patient first passes urine through the urethra, but this subsides on the following day." This was noted in a number of cases.

For the whole series the nineteenth day was the average on which micturition commenced. In 60 per cent spontaneous micturition commenced by the twenty-first day. In 25 per cent spontaneous micturition was delayed

until some time during the fourth week. In 14 per cent there was no micturition until an in-dwelling catheter had been used. One case was as late as the thirty-fourth day in commencing, and then only after an in-dwelling catheter had been employed. In two cases micturition was established on the day following the removal of the suprapubic drain.

In backward cases the use of the in-dwelling catheter undoubtedly helped to establish the habit. In one, however, it was not successful until after the use of the catheter for the second time. It was demonstrated that the slow return of micturition was one of the most important factors in connection with delay in closure of the fistula. It must be obvious that the final closure is impossible until micturition is re-established. Moreover, acute retention occurred in 2 cases whose fistulæ suddenly closed before micturition had commenced. They were treated with the in-dwelling catheter. With regard to the causes of the late onset of micturition, it was found that in 2 cases some obstruction to the passage of a sound was manifested between the prostatic cavity and the bladder. In about 90 per cent of those cases, however, with micturition delayed towards the end of the fourth week, there was one or other of the two following factors present in the history: (1) Symptoms of enlarged prostate for several years; (2) Marked chronic retention of recent origin. The inference is that loss of tone of bladder muscle from chronic retention was the chief cause of delay.

6. **The Time of Removal of the Suprapubic Drain.**—In 7 of the earliest cases of the series the drainage tube was removed on the seventh day. The motive for early removal was the hope of thereby shortening the convalescence. The average number of days before final closure of the fistulæ in these cases was twenty-eight, whereas the average for a similar number of cases whose tubes were removed on the tenth day, being treated at the same time, was twenty-one. Thus the object of early removal appears to have been defeated. It is an advantage to continue the drainage until the granulations in the prostatic cavity are well formed. There was a very striking tendency to unsteadiness in the temperature in those cases where early removal was practised. Freyer[1] points out that patients who pass urine early in the convalescence often do badly.

7. **Secondary Hæmorrhage.**—This occurred in 4 cases, about 6 per cent. In each case, owing to the state of the urinary tract, the patient was a poor subject for operation. The bleeding occurred at varying periods between the eleventh and the twentieth days. In no case was the hæmorrhage so severe as to cause any real anxiety. Each was treated by displacing the clots from the bladder, with irrigation by Janet's method, after dilating the fistula so that the large-size tube could be replaced, and this was left in position until all signs of bleeding had ceased. It is important to recognize that to deal effectively with this complication the bladder must be emptied of clot; otherwise the contractions set up by its presence will cause the hæmorrhage to continue. Hæmostatic serum and morphia hypodermically were useful aids.

8. **Malignant Cases.**—There were 7 of these—10 per cent of the total. All were early and recognized clinically before operation except one, and all offered some prospect of relief by prostatectomy. In 2 cases the enucleation

was accomplished with the forefinger. With the other 5 the removal was only completed with sharp dissection, after placing the patient in the Trendelenburg position. The average number of days in closing for 6 of these was twenty-one; the other case was discharged with a permanent suprapubic drain. Although the 6 cases closed so readily, 5 of them reported back to hospital within a few months with the fistula re-opened.

SUMMARY.

1. Too early removal of the suprapubic drain, by diminishing the drainage too soon, tends to delay convalescence.

2. Rapid closure of the fistula is not always desirable.

3. Closure of the suprapubic fistula without an in-dwelling catheter should be the aim for all cases. This was accomplished in about 52 per cent of the cases, and in 38 per cent by the twenty-eighth day of convalescence.

4. The in-dwelling catheter is necessary in a large proportion of cases to avoid an unduly protracted convalescence. It was employed in about 48 per cent.

5. The in-dwelling catheter does not help the fistula to close if used too soon; it increases the amount of sepsis present if left in too long. No complication arose from its use for three successive days in any case of this series.

6. In a considerable majority (about 66 per cent) the fistulæ were finally closed, either with or without the aid of an in-dwelling catheter, by the end of the fourth week.

7. In the remaining cases, the chief causes of delay in closure were: (*a*) Complications preventing the use of an in-dwelling catheter, such as acute epididymitis and pyelonephritis; (*b*) Delayed onset of spontaneous micturition, most commonly in cases of previous chronic retention; (*c*) Long-standing suprapubic fistulæ in two-stage prostatectomy cases; (*d*) A shelf of mucous membrane between the bladder and the prostatic cavity in some cases after Freyer's operation of prostatectomy.

8. Secondary hæmorrhage is not, as a rule, a serious complication, and can be readily controlled without operative interference.

9. The fistulæ in malignant cases may close very readily following suprapubic prostatectomy, but tend to re-open within a few months.

I have to thank the staff of St. Peter's Hospital for permission to publish these notes.

REFERENCES.

[1] FREYER, *Enlargement of the Prostate*, 1920, Baillière, Tindall & Cox.
[2] JANET, "Technique des Lavages sans Sonde de l'Urètre", *Jour. d'Urol. méd. et chir.*
[3] WHITE, WINSBURY, "Epididymitis and Suprapubic Prostatectomy: a Study of 50 Cases", *Lancet*, 1922, i. 321.
[4] DEAVER, "After-care in Suprapubic Prostatectomy", *Amer. Jour. Surg.*, 1920, July.
[5] MARION, *Traité d'Urologie*, 1921, ii, 1024, Masson et Cie.
[6] THOMSON-WALKER, SIR JOHN, "Hæmorrhage and Post-operative Obstruction in Suprapubic Prostatectomy; an Open Operation for their Prevention", *Brit. Jour. Surg.*, 1920, April, 525.
[7] THOMSON-WALKER, SIR JOHN, *Genito-urinary Surgery*, 1914, 712, Cassell & Co. Ltd.

SHORT NOTES OF RARE OR OBSCURE CASES

ACUTE HÆMORRHAGIC PANCREATITIS: A CASE PRESENTING CERTAIN UNUSUAL FEATURES.

By A. G. TIMBRELL FISHER, London.

The occurrence of more than one attack of acute hæmorrhagic pancreatitis in the same individual must be a rare condition. The case described below, in which two attacks occurred—separated by an interval of ten months—both of which were treated by successful operation, would appear to be of such extreme rarity as to justify detailed description.

W. H., age 58, a lighterman, was admitted to the Seamen's (Dreadnought) Hospital, Greenwich, on March 6, 1923, complaining of severe abdominal pain. He stated that he went to bed feeling perfectly well, but that at 8 o'clock the next morning he was seized with violent pain in the upper part of the abdomen shortly after awaking. The pain was agonizing, and sharp or stabbing in nature; it was continuous, although marked by fluctuations, and remained fairly localized. He made no complaint of pain in the back. He vomited once soon after the commencement of the pain, and there were two movements of the bowels within half an hour of its onset, since which there had been no action. He was seen by his doctor, who ordered his removal to hospital.

Previous History.—The patient, until his recent series of admissions to hospital, had enjoyed excellent health, and was of temperate habits. His digestion had been good, and he had never been troubled with constipation. He had never been jaundiced or suffered from colic. He had been an in-patient at the hospital on three separate occasions in the preceding year:—

1. He was admitted on Nov. 8, 1921, with a fracture of the neck of the right femur, and discharged on Jan. 6, 1922, with good function.

2. On May 5, 1922, he was operated upon by my colleague, Mr. E. T. C. Milligan,* for acute hæmorrhagic pancreatitis, of which he presented the classical signs and symptoms. Violent epigastric pain commenced at 10 p.m. on May 4, and the operation was performed at 8.30 p.m. on the following day. At operation, Mr. Milligan found much dark blood in the general peritoneal cavity, particularly in the right kidney pouch. The lesser peritoneal sac was also full of blood. The pancreas was swollen and dark purple in colour, and

* I am indebted to Mr. Milligan for the notes of the condition found at operation.

the body of the gland contained extravasated blood. There was fat necrosis of the omentum. A tube was introduced below the stomach into the lesser sac. At first there was a blood-stained discharge ; later, this became purulent, and continued for some time. Some digestion of the abdominal wall occurred, which, however, ceased on the application of dilute acetic acid dressings. A pathological investigation of the fæces on May 11 revealed no excess of free fats, but fatty acid and soap crystals were present. A twenty-four-hour specimen of the urine showed the presence of 5 units of diastase. He made a good recovery, and left the hospital on July 13.

3. He was next admitted on Oct. 19, 1922, with a simple transverse fracture of the right patella, with but little separation of the fragments. This was treated by non-operative methods, and he left hospital to attend as an out-patient on Nov. 27. He made a good functional recovery, and as I signed his discharge certificate I expressed the hope that his series of admissions to hospital had now come to an end. But the Fates ordained otherwise, and he was admitted, as already mentioned, with a second attack of acute hæmorrhagic pancreatitis.

Condition on Admission.—The patient, a somewhat stout, well-nourished middle-aged man, presented the signs and symptoms of an acute abdominal catastrophe of the upper abdominal type. A considerable degree of collapse was present ; the skin was cold and clammy, the pulse rapid and of poor volume, and the temperature subnormal. A striking feature was a peculiar leaden colour of the skin, with a definite cyanotic tinge of the lips and face. He was restless and complained much of thirst. Pain was extremely severe, and situated in the epigastric region and in the upper left quadrant of the umbilical region, the pain being more marked at the latter site. As already mentioned, it remained localized and did not penetrate to the back, but was subject to exacerbations. The abdomen was generally distended, the distention being more marked above the umbilicus. Abdominal tenderness was marked in the epigastric region, but the abdomen moved slightly on respiration, and there was a complete absence of the board-like rigidity which is usually associated with such acute upper abdominal catastrophes as ruptured gastric or duodenal ulcer. He was troubled with a cough, and the expectoration was ' rusty ' in appearance. Examination of the chest revealed dullness to percussion of both lung bases, with moist crepitations. The presence of cyanosis and the absence of board-like rigidity, in conjunction with the other signs and symptoms—although I was at the time unacquainted with the patient's previous history—influenced me considerably in arriving at a diagnosis of acute hæmorrhagic pancreatitis, although it was clear that serious trouble existed above the diaphragm in addition.

Unfortunately, it is considered by some that the presence of a most unyielding type of *generalized* abdominal rigidity is characteristic of acute pancreatitis ; but this is not borne out by my personal observations of this and other cases. Extreme rigidity, localized in the epigastric region, with a somewhat flaccid abdominal wall elsewhere, may, however, occur. Moynihan[1] states : " The abdomen, when examined early, presents the most indomitable rigidity and some fullness in the upper part : the remaining parts may be quite soft and flaccid, yielding readily to the hand, or they may be

held with some degree of firmness." Dick, in his thoughtful M.D. thesis,[2] has emphasized, from his experience of six cases in general practice, this characteristic absence of true peritonitic rigidity.

The presence of cyanosis as a characteristic feature was first noticed by Halsted.[3] It has been suggested that this cyanosis is toxic in nature, and due to digestion of the pancreas by its own escaped secretions. I venture to ask tentatively whether it may not be due, either in whole or part, to interference with respiration and consequent deficient oxygenation of the blood, which may occur either reflexly or from mechanical interference with respiration. It is rash to argue from one case, but the phenomena noticed at the operation related below are certainly suggestive. In the present case the condition of the lungs must also be taken into consideration as a possible contributory factor in the causation of cyanosis.

Operation.—The abdomen was opened through a para-median rectus-displacing exposure in the left side of the epigastric and umbilical regions, at 5 p.m. on the day of admission—i.e., nine hours after the onset of symptoms. The abdominal cavity was full of very dark blood, but no obvious areas of fat necrosis could be seen. The bowel, both small and large, was congested, and some paralytic distention was present. The mesentery at its root was distended with blood-clot, as was also the lesser sac of the peritoneum. Access to the latter was obtained by a vertical incision in the great omentum immediately below the greater curvature of the stomach. A large quantity of blood-clot was removed from the lesser sac, and then a remarkable alteration in the patient's colour and general condition, and in the character and volume of the pulse, became almost immediately evident. On further investigation the pancreas was found to be much enlarged by hæmorrhage; and in the peritoneum of the posterior wall of the lesser sac, which clothes the anterior surface of the body of the pancreas, was a ragged opening through which the forefinger could be introduced, a gush of blood following the procedure. This opening was enlarged with the scalpel, and as much as possible of the friable blood-clot in the vicinity and in the substance of the pancreas was removed. A large rubber drainage-tube was introduced through the opening into the pancreas and the abdomen closed. The patient's condition at the end of the operation was markedly improved—his pulse was of better quality, and a striking difference in the colour could be noticed.

After-history.—For many days there was a copious discharge of dark blood from the drainage tube, and the abdomen remained distended, with persistent dullness in the right lumbar and iliac regions. This dullness was evidently due to blood from the lesser sac, which had escaped via the foramen of Winslow and taken this course owing to the mesenteric attachment. The 'rusty' tenacious sputum persisted for a few days and then became clear, and *pari passu* the lung condition gradually cleared up. The discharge of blood from the abdominal wound continued very free, and a certain amount of abdominal distention still persisted, while the temperature remained intermittent. About a fortnight after the operation, bile-stained pus began to escape from the wound, and some digestion of the abdominal wall occurred, which was satisfactorily treated by means of acetic-acid dressings. Two very large sloughs of pancreatic substance escaped from the wound four weeks after

the operation, after which the discharge promptly ceased. His condition rapidly improved, and he was sent to a convalescent home.

The occurrence of suppuration followed by sloughing of pancreatic substance points to the fact that the three forms of acute pancreatitis described by Fitz—viz., hæmorrhagic, suppurative, and gangrenous—may, as Moynihan points out, be different stages of the same disease.

Commentary.—The experience of this case has strengthened in my mind the impression I had already obtained, that acute pancreatitis presents a definite clinical picture, and that diagnosis should be possible by attention to certain distinct features. It seems clear that many text-books, instead of endeavouring to point out its peculiar distinguishing features, are too apt to attempt to bring it into line with other abdominal catastrophes.

Most of us are familiar with the dicta of Fitz,[4] a pioneer upon this subject, who wrote: "Acute pancreatitis is to be suspected when a previously healthy person or sufferer from occasional attacks of indigestion is suddenly seized with violent pain in the epigastrium, followed by vomiting and collapse, and, in the course of twenty-four hours, by a circumscribed epigastric swelling, tympanitic or resistant, with slight rise of temperature." And again: "The symptoms are essentially those of a peritonitis beginning in the epigastrium and occurring suddenly during ordinary health without obvious cause."

Let us analyse these statements a little more closely, and inquire whether recent advances have not provided us with a somewhat clearer picture. In the first place, it may be stated that nowadays it is unlikely that anyone would wait twenty-four hours, in the presence of the acute symptoms mentioned, for the occurrence of 'circumscribed swelling' in order to confirm a possible diagnosis of acute hæmorrhagic pancreatitis. The remainder of the clinical picture applies with equal accuracy to a number of acute upper abdominal conditions, and is by no means pathognomonic of acute hæmorrhagic pancreatitis. In my experience, the collapse, pallor or cyanosis, restlessess, thirst, increasing abdominal dullness and distention, and absence of board-like rigidity, point to intra-abdominal hæmorrhage rather than to peritonitis. The violent nature of the pain is, of course, explicable by the position of the primary source of the hæmorrhage, which causes extreme stretching of the parietal peritoneum covering the pancreas; and the profound collapse by the proximity of the solar plexus. I think, therefore, we are in a position to modify the otherwise excellent description of Fitz and to state: "Acute pancreatitis is almost certainly present when a person is suddenly seized with violent pain in the epigastrium, followed by vomiting, severe collapse, and cyanosis, and by the symptoms of intra-abdominal hæmorrhage, unassociated with typical peritonitic rigidity."

Second Attacks of Acute Hæmorrhagic Pancreatitis.—The occurrence of two attacks must be a very rare condition. I have been able to find a record of only one such case, and in this death occurred during the second attack. The case is recorded by Dick[2] as follows:—

"A. B., male, age 44, a publican, was well known to me prior to his present illness, and was definitely alcoholic in his habits. I was called to see him on April 12, 1905, and found him in bed suffering from epigastric pain, obviously

very severe. The pain was constant, but with paroxysmal exacerbations, and its severity was of a degree altogether exceptional; he showed signs of profound shock, there was general clammy pallor, and his pulse was hardly to be felt. Temperature 97·4°. On inspection of the abdomen there was some limitation of movement on respiration, but this was not pronounced. On palpation there was no well-marked general tenderness in the epigastrium, and no definitely localized tender spot; steady pressure tended to relieve the pain. The condition appeared to me to be unusually severe biliary colic, although I had never from previous experience known him to be harbouring gall-stones. A full dose (½ gr.) of morphia was given hypodermically, but to my great surprise failed to give any relief; ¼ gr. was therefore given an hour later, again without relief; the pain and collapse were unaffected.

"On the second day of the illness the condition was the same, with certain symptoms added—notably great restlessness and intense thirst. The former was very noticeable, and it was an altogether unusual thing to see a man so profoundly ill—with no pulse at the wrist, and collapsed—attempting to get out of bed apparently with the idea of getting relief from pain by change of posture. Once again morphia failed to relieve, although repeated until 1 gr. had been given hypodermically in twenty-four hours, the initial dose being again ¼ gr. There was considerable nausea, with occasionally small quantities of bilious vomit, but vomiting was at no time a striking symptom.

"On the third day there was to be observed some circumscribed swelling in the epigastric region above the umbilicus. There was complete constipation, but flatus could be passed, and there were not present the signs of intestinal obstruction. By now it was borne in upon me that I was witnessing a combination of clinical symptoms which I had never previously seen, and constituting to me a new disease.

On the fourth day the severity of the symptoms as regards pain and collapse had slightly abated; the pulse returned at the wrist, and was feeble, rapid (112), and of low tension. Temperature 98·8°. Still no action of the bowels.

"Fifth day and afterwards: Improvement was gradually maintained; the bowels were moved on the seventh day, a large greenish slimy evacuation.

"On the tenth day he was able to be out of bed, and, although very weak, was comparatively well.

"This initial attack lasted from April 12 to 21.

"Subsequent History from April 20 to June 21.—After April 20 he gradually recovered strength, and resumed his ordinary occupation. The only persisting symptom which I noted was '*pain in the back*', brought on by stooping while working in the garden. The pain was in the lower dorsal region, and not acute—more of a dull ache—and always brought on by continuous stooping. On June 20 I was again summoned, and found him in acute pain and collapse, which lasted for twenty-four hours and terminated fatally, all palliative treatment being again unavailing; the symptoms were identical with those occurring on April 12.

"Post-mortem Examination.—The abdomen only was investigated. There was no general peritonitis. The liver showed signs of the early fatty stage of cirrhosis. The gall-bladder contained no gall-stones, nor were any to be found in the cystic and common ducts. The fat in the root of the mesentery and omentum was here and there converted into soft lumps of the consistency of butter. The lesser sac of the peritoneum was distended and full of turbid chocolate-coloured fluid, and floating free in this fluid were dark lumps of old and recent blood-clot, and tougher masses consisting of gangrenous pancreatic tissue. The pancreas was disorganized; parts of it were soft and pulpy, other parts tough and indurated; there were areas showing old and recent hæmorrhage.

"*Microscopical Examination of the Pancreas.*—Difficulty was experienced in securing a portion of the gland which was not hopelessly necrotic, for microscopical examination. On examination, however, early and later inflammatory changes were found: the former evidenced by small-celled infiltration, and the latter by overgrowth of fibrous tissue. Crystals of hæmatin were distributed throughout the specimen. The glandular element showed great disintegration, part of which may have been due to post-mortem changes."

Etiology.—Controversy raged for many years around the question as to whether the pancreatic hæmorrhage was primary, or whether some inflammatory condition of the pancreas preceded and caused the hæmorrhage. The consensus of opinion in modern times appears to be that both modes of origin may occur. Fitz certainly inclined to the view that hæmorrhage might be directly causative. Mayo Robson considers that the ultra-acute type is probably due to a primary hæmorrhage, whereas, in the cases with somewhat less acute symptoms, the hæmorrhage is preceded by inflammatory processes in the pancreas. Everyone is familiar with Opie's classical experiments. Unfortunately, the question is still in the realm of hypothesis, and cannot yet be said to rest upon a scientific foundation. Possibly the primary hæmorrhage factor has been exaggerated. Certainly we know that the hæmorrhages that occur in the pancreas after trauma, in certain abnormal blood conditions, in infectious diseases, heart disease, and atheroma, are not necessarily followed by acute pancreatitis.

To return to the case under consideration: the absence of gall-stones and of any symptoms suggesting an infective condition of the alimentary tract is of interest; yet the presence of the coexistent infective foci in the lung bases is of still greater import, and strongly suggests that the pancreatitis was also infective in nature.

It is interesting to note, that in spite of the necrosis of a large part of the body of the pancreas that undoubtedly occurred, the patient presents no signs of pancreatic insufficiency.

REFERENCES.

[1] Moynihan, Sir Berkeley, *Abdominal Operations*, 3rd. ed., ii, 396.
[2] Dick, R. J., "Acute Pancreatitis", *Edin. Med. Jour.*, 1910, Sept.
[3] Halsted, *Johns Hopkins Hosp. Bull.*, 1901, Nos. 121, 122, 123.
[4] Fitz, *N. Y. Med. Jour.*, 1889.

A CASE OF JEJUNAL DIVERTICULA.

By L. R. BRAITHWAITE, Leeds.

The following case presents a condition of such rarity that it merits publication.

J. H., age 54, was brought to my notice by Drs. Taylor and Harrowell on March 3, 1917, as a case of chronic intestinal obstruction probably due to old tuberculous peritonitis. I diagnosed the condition as one of atonic dyspepsia, advising lavage and an X-ray examination. The following is a copy of Dr. Rowden's report dated March 8, 1917:—

X-ray Examination.—*Stomach*: "Is somewhat dilated. Tone good. Outline regular. Peristalsis active, and the waves are seen to pass along greater and lesser curvatures right up to the pylorus. It is about one-half empty in one and a half hours, and completely so three hours after the opaque meal. No X-ray evidence of gastric ulcer. No pyloric obstruction.

"*Duodenum*: No food lodges. Pyloric cap not visible. No evidence of duodenal ulcer.

"*Small Intestine*: In several places (three or four) the food accumulates in sacs or pouches in the region a little to the left of the umbilicus. These are probably in the jejunum or proximal portion of the ileum. Beyond these pouches there is partial obstruction, probably due to bands, or pressure of enlarged mesenteric glands. In spite of these 'obstructions' some of the meal has arrived at the cæcum in five hours."

I did not see the patient again until Oct. 31, 1922, and at that time he presented very definite physical signs. There was obvious small-intestine peristalsis, confined to the left half of the abdomen. His symptoms led me to

FIG. 135.—X-ray examination. Result 24 hours after opaque meal.

suppose him to be suffering from tuberculous peritonitis, with subsequent adhesions and multiple obstructions in the upper half of the small intestine. I admitted him to the General Infirmary at Leeds with this diagnosis, and the following is a copy of his case-history and operation:—

HISTORY.—Patient admitted for pain and nausea after food. Present condition has lasted six years definitely. The patient on eating food feels a foul taste in the mouth immediately after the first mouthful. This is followed by formation of much wind, which is eventually passed as flatus or by the mouth. Moreover, there is a feeling of food retention in the lower part of the abdomen, which is relieved generally by taking another meal. Vomiting

used to take place three or four times a week, two or three hours after food, the vomit consisting of the food taken before, yellow in colour and acid in taste. Eighteen months ago hæmorrhage by the mouth occurred, consisting of some five pints of unaltered blood. It followed an ordinary vomit. After this the vomiting ceased. The patient has lost two stone in weight during the six years; he has generally been constipated. Micturition normal.

On Examination.—The abdomen is distended. Wall is somewhat tense. No definite pain on pressure. Marked visible peristalsis, confined to left half of abdomen.

Operation, Nov. 9, 1922.—Right rectus incision. The upper third of small intestine showed numerous (about fifty) diverticula, along its mesenteric

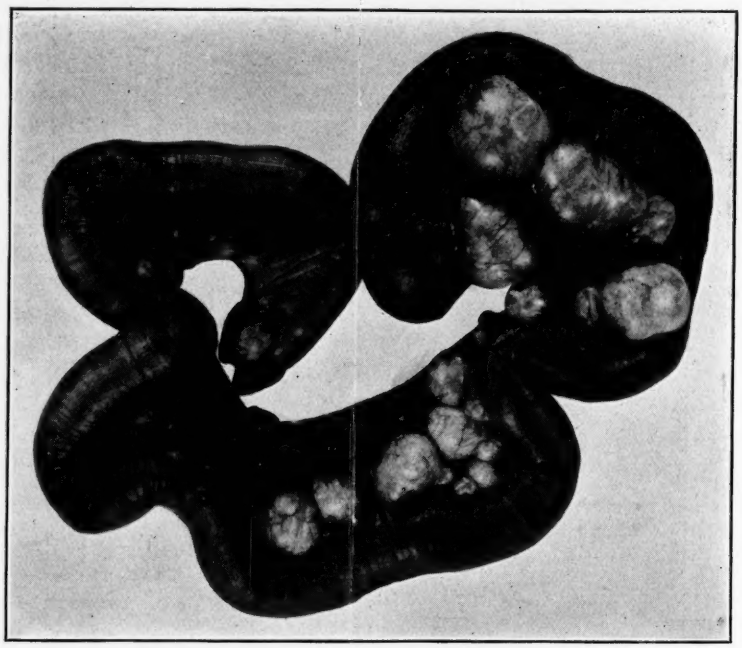


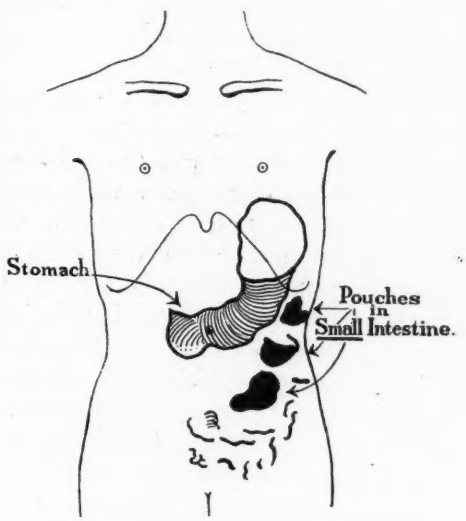
Fig. 136.—Showing the portion of intestine removed at the second operation.

border, but bulging free into the peritoneal cavity. The largest was larger than a Tangerine orange; the smallest the size of a pea. Large diverticula showed inflammatory changes with adhesions. Some seemed to be almost on the point of perforation, the walls being white and extremely thin, like tissue paper. The first diverticulum was at the duodenojejunal flexure. The upper few feet of the small intestine were hypertrophied and dilated. Owing to the presence of the uppermost diverticulum actually at the flexure, excision was thought to be inadvisable. A lateral anastomosis was made between the antimesenteric border of the upper end of the jejunum and the intestine below the diverticula. Numerous enlarged glands were found in the mesentery of the jejunum; one was removed for examination. He made an excellent recovery from the operation, and on Nov. 20 an X-ray examination was made by Dr. Scargill, the result of which was inconclusive. The following

is the report by Dr. Scargill: "(1) Three hours after barium meal; (2) Fifteen minutes after barium meal. There is a retention of barium in the small intestine after most of it has passed into the lower coils, but it is not possible to radiograph whilst the jejunum is filled."

The result of the anastomosis was not good. He was relieved in some measure, but began to suffer from attacks of pain, vomiting, and abdominal distention, which indicated to my mind the filling of the diseased portion of the intestine by gas, and traction of the distended loop on the duodeno-jejunal flexure, giving rise to mechanical obstruction.

An X-ray examination was now carried out by Dr. Rowden. *Fig.* 135 represents the condition found at this time, and shows with extraordinary clarity many of the diverticula apparently filled with bismuth. The horizontal line made by the bismuth as it lay in the diverticula is particularly well shown. It is of interest that most of the bismuth found its way immediately through the new anastomosis and into the lower portion of the small intestine. Only a small portion escaped along the natural path into the diseased portion of bowel.



FIG, 137. — Condition about 1 hour after opaque meal. Copied from the diagram made by Dr. Rowden in July, 1916.

SECOND OPERATION. — He was re-admitted to the Infirmary, and a second operation was performed on March 28, 1923. The uppermost diverticulum, which lay at the duodenojejunal flexure, was infolded. The portion of intestine infected by disease, amounting to about four feet, was then excised immediately below the existing anastomosis, and the divided ends were closed. *Fig.* 136 is a photograph of the specimen. He made an excellent recovery and has remained well since.

The case presents features of great interest with respect to the radiographic findings. It is remarkable to note that so early as July, 1916, on examination made by Dr. Rowden, a definite diagnosis of pouching in the small intestine was made, and *Fig.* 137 is an exact copy of Dr. Rowden's diagram made on that occasion.

With regard to the operative procedure adopted, the real difficulty in the case was presented by the uppermost diverticulum. It was realized that to remove this diverticulum and the bowel contiguous to it would leave such a short end with which to make an anastomosis that the certain union of it would be in doubt. The only procedure to be adopted would have been to close the upper end of the jejunum and do a gastro-enterostomy. In the year 1914 I published in the BRITISH JOURNAL OF SURGERY three cases in which this procedure was adopted, but for a different condition.

Eventually it was decided that the uppermost diverticulum might possibly be treated by infolding, rather than by excision, leaving sufficient

intestine along its antimesenteric border with which to do an anastomosis. It is rather too early to say with any certainty whether the infolding operation as adopted can be of any permanent value. It is to be supposed that there will be some danger of an expansion of this diverticulum, with a chance of perforation.

Then with regard to the symptomatology. One would have anticipated that, owing to the filling of the multiple diverticula and traction therefrom, a state of acute intestinal obstruction would have been inevitable. The absence of this is possibly accounted for by the fact that the openings of the diverticula into the intestine were very small, and most of the contents would pass along the canal. It would appear certain that after the first operation his symptoms were due purely to traction from a filling of the isolated loop with its diverticula.

It was considered that a personal narrative given by the patient himself would be of considerable interest. The following is a copy of his history as he described it :—

"As far back as I can remember I have suffered more or less from dyspepsia. When 18 years old I had an accident in the cricket field, my right knee being put out. Until 35 years of age I suffered from periodical attacks of indigestion, with much flatulence and pain in the stomach and bowels, and much vomiting. Often these attacks were accompanied with synovitis in the knee, which caused great pain and inconvenience. From 35 to 45 years of age I was entirely free from synovitis, and had very little indigestion, my general health being very good.

"In 1915 and during the War period I spent much of my time in London. The air raids, which were numerous, had an effect on my general health, the indigestion again appearing, and, in due course, the synovitis. After a meal I had an hour fairly comfortable, then a swelling of the body took place, with much flatulence in the stomach and intestines. The weird noises made it very uncomfortable to sit in a room with other people, and often I had to retire to my own room until these noises subsided, which used to be in about two hours. After much retching the wind passed off in the natural downward way—then relief. When, however, the wind did not pass naturally, violent sickness ensued.

"In 1916 I was examined by X rays (Dr. Rowden—*see Fig.* 137). In 1917 I experienced an obnoxious taste every time I had a meal. This taste generally subsided by the time the meal was finished, but eventually it was the cause of much trouble, and was diagnosed by a physician as septic poisoning from some cause which could not be found. In January, 1920, after a lunch of ordinary size, I had a violent attack of sickness followed by internal hæmorrhage, parting with about five pints of blood. Previous to this occasion I felt to be in good health. My teeth were suspected, and I had them all extracted in March, 1922. The taste was worse than ever afterwards. An operation was later advised, and took place in the Leeds General Infirmary in November, 1922 (*see* report). After this operation the bad taste disappeared, but other troubles, chiefly vomiting, presented themselves. A second operation was advised, and was carried out in March, 1923. To date, May 10, 1923, operation is working quite satisfactorily."

AN UNUSUAL CAUSE OF DEATH IN ACUTE APPENDICITIS.

By C. HAMILTON WHITEFORD, Plymouth.

On Feb. 24, 1923, a seaman, age 21, was admitted to the Plymouth Infirmary with a history of ten days' illness—intermittent abdominal pain—while at sea. The ship carried no doctor, and details of this period of the illness were not available. The medical man who first saw the patient found no symptoms of acute abdominal disease.

Seen by the writer on the 11th day of the illness, the condition was as follows: Patient sallow and rather thin, with cough and râles in both lungs. Respiration 26, pulse 92, temperature 100°. He complains of intermitting abdominal pain in right side of abdomen and epigastrium. He states that he has lost flesh, and always has a cough, and that the abdominal discomfort commenced about ten days ago. There had been no previous abdominal illness. The abdomen was retracted, not boarded, and slightly tender along the ascending colon. No tumour was palpable. Per rectum there was neither tenderness nor tumour. Indications for immediate operation were not obvious, and the opinion was expressed that the symptoms could be explained by a pulmonary lesion (possibly tuberculous) with pain referred to the abdomen.

On the 15th day the leucocyte count was 17,000, respirations 30, pulse 90, temperature 99°. Abdominal tenderness as on admission. There had been no vomiting, and the bowels had acted after enemata. It was now ascertained that on the 10th day there had been a rigor lasting ten minutes, and the patient stated that, during his illness at sea, he had several 'attacks of shivering.'

On the 16th day the respirations were 24, pulse 70, temperature 98·4°.

Operation.—Under the anæsthetic, a deep-seated resistance was palpable in the outer part of the right iliac fossa. The cæcum was exposed from its right side. The general peritoneal cavity was not shut off by adhesions, and was packed off with gauze. A tumour the size of a hen's egg was found lying to the right of and behind the cæcum, which formed part of the front wall of the tumour. The abscess was opened with the finger, and one ounce of thick greenish-yellow pus spurted out. The pus was followed, at once, by a stream of dark blood, which welled up from the abscess cavity. Half a pint of blood escaped. The abscess cavity was 'bipped' and packed with gauze, which stopped the bleeding. A half-inch rubber tube was passed down to the gauze in the cavity. The appendix was neither seen nor felt. The gauze removed from the peritoneal cavity contained no blood.

Twenty hours after operation the respirations were 26, pulse 94, temperature 99°. The gauze was removed from the abscess cavity, and the tube was shortened. There was no recurrence of bleeding, and no more gauze was inserted. Twenty-seven hours after operation, the patient, while talking to his brother, collapsed, and died in five minutes. There was no bleeding from the wound.

Autopsy.—Permission was obtained only for examination of the abdomen through the incision. The abdomen was full of fluid blood and clots, none of which were adherent to or incorporated with the surfaces of the viscera.

The walls of the abscess were infiltrated with clot, and no opening could be demonstrated in the iliac veins. The abscess lay between the cæcum and the pelvic brim. The rest of the abdomen showed neither adhesions nor peritonitis. The appendix, three inches in length and gangrenous throughout, was still attached to the cæcum, which was not perforated.

Comment.—The sequence of events appears to have been as follows: Acute inflammation and gangrene of a retrocæcal appendix. Spread of infection to a vein (? external iliac) in the wall of the abscess. Septic thrombosis of the vein, with detachment of small emboli, which lodged in the lungs. Evacuation of the pus left the vein unsupported, and the vessel ruptured into the abscess cavity. Gauze pressure closed the opening in the vein and permitted formation of a thrombus. The thrombus became softened or displaced, and blood poured from the vein into the peritoneum via the abscess cavity.

Disproportion between the respiration and pulse rates was a marked feature throughout, and may have been due to lodgement in the lungs of small septic emboli.

J. D. Malcolm (*Surgery, Gynecology, and Obstetrics*, Nov. 1908, p. 529) mentions four similar cases. Of these, two recovered after ligation of the external iliac artery, one recovered after the abscess cavity was packed with gauze, and one "rapidly succumbed".

PERFORATION OF A MECKEL'S DIVERTICULUM: OPERATION: RECOVERY.

By DOUGLAS DREW, London.

This case appears worthy of record, as the child was operated upon at the age of 10 months by Mr. F. J. Steward for an intussusception of a Meckel's diverticulum; the intussusception was reduced and the abdomen closed, as the age of the child and its serious condition did not permit of the removal of the diverticulum.

Mr. Steward has kindly supplied the following note:—

I operated upon this patient at the age of 10 months for intussusception. On opening the abdomen the intussusception was found to commence at the junction of a Meckel's diverticulum with the ileum. The diverticulum was about 2½ in. long, and had advanced into the ileum a distance of about 3 in. The parts were considerably congested, and reduction was difficult, but was accomplished at the expense of several tears in the peritoneal coat of the ileum. The question of resection of the diverticulum was then considered, but was abandoned because the condition of the parts was such that resection of a considerable length of ileum with the diverticulum would have been necessary, and I did not think it wise to undertake this, as the child was then somewhat collapsed. Recovery took place without incident.

No further trouble occurred until the boy was 9¼ years old, when I was called to see him in consultation (August, 1922). He then had acute abdominal symptoms suggesting acute appendicitis—sickness, abdominal

pain and tenderness, raised temperature, rapid pulse, and rigidity of the abdominal wall.

The question was discussed as to whether the trouble might be due to the diverticulum, and bearing this possibility in mind the abdomen was opened on the right of the umbilicus.

The transverse colon, small intestine, and omentum were adherent around the umbilicus. In separating the adhesions, a small collection of thick dark-green fluid resembling bile was evacuated. After the adhesions had been freed, the bowel was drawn outside and the omentum separated from the diverticulum, which it enveloped. The diverticulum was flask-shaped, about 1½ in. long by 1 in. wide, with a narrowed neck where it joined the bowel (*Fig.* 138); this was clamped flush with the bowel, a catgut ligature applied, and the stump buried with a purse-string suture. There was evidence of early peritonitis, with a quantity of slightly turbid and bile-stained fluid in the flanks and the pelvis, which was carefully sponged away; the appendix, which was normal, was removed. A short coil of the small intestine, about six inches, in the middle of which was the attachment of the diverticulum, was very considerably hypertrophied, the limits of the hypertrophy being quite sharply defined. I decided against inserting drainage tubes, as the operation was performed within twenty-four hours of the onset of symptoms, and for the reason that in perforated duodenal ulcer in the early stages it is not necessary, and the infectivity of the contents of the small intestine at the level of the diverticulum was not likely to be much greater than that of the duodenum.

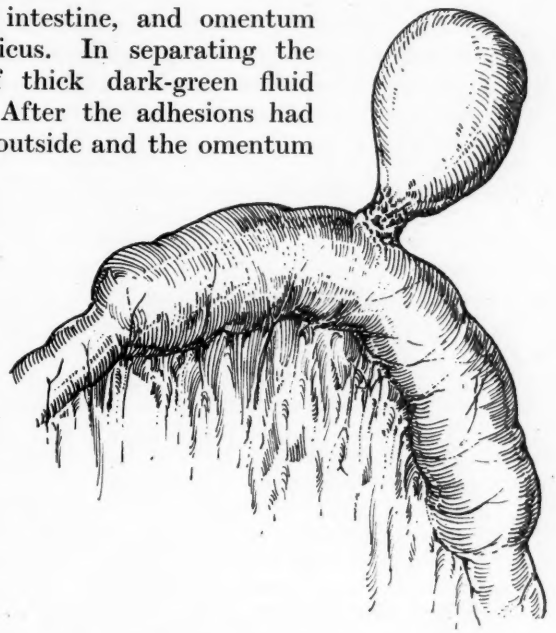


Fig. 138. — Meckel's diverticulum proceeding from the middle of a portion of hypertrophied intestine.

It will be noted that no mention has been made of a perforation, for the reason that none could be discovered. On careful examination of the diverticulum after its removal, the interior of the body of the flask was empty, and its mucous membrane did not show any sign of inflammation, but that of the neck of the flask was red and inflamed; no perforation, however, could be found. In the absence of a microscopic perforation due to an agent such as an undiscovered fish bone, I think that the peritonitis arose from infection spreading from the inflamed part of the neck, and was possibly determined by the drag upon it by the hypertrophied part of the bowel.

The case, therefore, differs somewhat from a Mickel's diverticulitis, as the pouch was empty and the mucous lining was not inflamed except as above stated.

REVIEWS AND NOTICES OF BOOKS.

A Text-book of the Surgical Dyspepsias. By A. J. WALTON, M.S., M.B., B.Sc. (Lond.), F.R.C.S. (Eng.), Surgeon (with charge of Out-patients) London Hospital, late Surgeon Poplar, Greenwich, and Evelina Hospitals, late Hunterian Professor Royal College of Surgeons. 8vo. Pp. 728. 1923. London: Edward Arnold & Co. £2 2s. net.

IT is stimulating to find 'gall-stones' and 'chronic appendicitis' at last in a book entitled *The Surgical Dyspepsias*; not so much that these conditions are surgical, as that their inclusion emphasizes their symptomatology. Mr. Walton's excellent book fills an undoubted hiatus, in that it embraces almost all the surgical abdominal conditions associated with dyspepsia, and gives the most carefully selected extracts in regard to their etiology and pathology; it fills a place which could only be furnished otherwise by a perusal of at least a dozen works or monographs of 'specialists' in the various dyspepsias.

Chapters I and II are invaluable in that they recite in detail the surgical anatomy of the stomach, and give a very careful account of the methods of examination of a case of dyspepsia. In Chapters III to VII the cause, diagnosis, and treatment of gastric and duodenal ulcer are adequately dealt with in the light both of modern research and the experience of the greatest workers. Pyloric stenosis is discussed in Chapter VIII, gastric neoplasms and their treatment in Chapters IX and X. On p. 211 the author dismisses in eleven lines that by no means rare condition "chronic duodenal ileus", and suggests no method for its cure. Rare conditions, e.g., foreign bodies and injuries of the stomach, acute dilatation, volvulus, and intussusception are sufficiently described in Chapter XI.

It is in connection with the technique of gastric operations and the operative treatment of gastric lesions (Chapters XII, XIII, and XIV) that one would wish to make some criticism. The author seems to suffer from his anæsthetists. On p. 314 he says with reference to closure of the peritoneum and posterior sheath, "This will generally be found the most difficult part of the whole operation"; and on p. 315, "If the patient be struggling considerably". Surely this is not the usual experience of surgeons.

The technique of performing gastrojejunostomy varies in different surgical clinics, but rigid surgical cleanliness should be common to all. Mr. Walton, like so many other very brilliant and successful operators, is inclined to judge results rather on mortality than on the quality of the recovery. Surely before an incision is made into the lumen of the stomach and jejunum, some warning should be given that a potentially septic focus is about to be invaded, and some precaution be taken to avoid spread of the infective agent over the so far sterile pads.

In the treatment of gastric ulcer, pride of place is given to wedge resection, with a posterior gastro-enterostomy and occlusion of the pylorus. Numerous authorities are quoted to support this line of treatment, and Mr. Walton expresses himself as well satisfied with it. It is quite certain that this line of treatment is not nearly so successful as regards the late after-results in the hands of many others. Not only are symptoms likely to recur, but new ulcers to form, both in the site of the wedge resection and in the line of the gastrojejunal anastomosis. In addition, wedge resection cannot be considered sufficient for those cases, possibly not so rare as one may imagine, where malignant change is beginning to take place. It is for

partial gastrectomy one pleads, an easier operation and in skilled hands more rapidly performed, with a mortality little, if any, greater than that from simple gastrojejunostomy, with a quality of recovery and a permanency of freedom from all symptoms as satisfactory as is achieved in the treatment of duodenal ulcer.

Chapters XV to XVIII deal with gall-stones and their complications. Following a lucid chapter on the surgical anatomy of the liver and gall-bladder, the etiology and pathology of gall-stones are very well represented. One is pleased to find flatulent dyspepsia described as the earliest symptom of gall-stones, and a strong plea made for early surgical treatment. The author gives a perfectly fair discussion of cholecystotomy versus cholecystectomy, and decides definitely—and in our view rightly—in favour of the latter. He makes the very true remark that a probe cannot be passed along the cystic into the common duct, but suggests it is a simple matter after the gall-bladder is removed. One finds very frequently, however, that unless the cystic duct is divided *at* its junction with the common duct (not a good thing to do if one is to place a ligature or suture upon it afterwards), the stump of the cystic duct that is left has to be *slit up* into the common duct before exploration of the common duct can be accomplished. As in gastric so in gall-stones work, Mr. Walton errs in his technique. There is not nearly sufficient care in shutting off the surroundings from the potentially infective bile; and by advocating a drain into the right renal pouch after simple cholecystotomy he would seem to admit his own doubts.

Chapters XIX and XX are devoted to the pancreas. They are well written and quite sufficiently in detail for all practical purposes.

Chapters XXI and XXII are devoted to a consideration of visceral ptosis and appendix dyspepsia and their treatment. One could wish that less space were given to the former and more to the latter. It is surely notorious how unsuccessful the majority of operations for visceral ptosis are, however certain it is that the symptoms are real enough. One could have wished the author had laid more stress upon the belt or support associated with his name.

As a whole, one welcomes the book very warmly; it is certainly a fine compendium for the senior student; it will be exceedingly useful for those in general practice who desire to know exactly what surgery is doing for dyspepsia; but it is not nearly an adequate technical work to guide the steps of a young surgeon.

Mr. Shiell's drawings are clear enough, but some of them are crude. They do not suggest familiarity with the living subject; they compare unfavourably with the quality of work seen, for example, in the American books on similar subjects.

Die Knochenbrüche und ihre Behandlung: Ein Lehrbuch für Studierende und Aerzte. By Dr. Med. Hermann Matti, Privat docent für Chirurgie an der Universität und Chirurg am Jennerspital in Bern. Royal 8vo. In two volumes. 1918 and 1922. Berlin: Julius Springer.

Vol. I.—General Principles of Fractures and their Treatment. Pp. 395, with 420 illustrations. Unbound, 20s. Bound, 23s. 8d.

Vol. II.—Special Fractures, including Fractures of the Skull and Spine, together with their Complications. Pp. 985 + xi, with 1054 illustrations. Unbound, 50s. Bound, 53s. 4d.

There can be no doubt that this work represents a most important and valuable addition to the systematic description of fractures. The first volume deals with general principles. The opening chapters describe the incidence of fractures with relation to age, sex, and occupation, the architecture of bones, and the mechanics of fractures with relation to bony structure and the breaking force. These sections are clearly written and excellently illustrated both by diagrams and skiagrams.

The next section deals with the pathological anatomy and physiology of recent fractures, and is accompanied by very well chosen photographs of typical museum specimens. There is a special chapter on *x*-ray technique in fracture examination.

The remainder of the volume is chiefly occupied by a description and discussion

of the general principles of fracture treatment. Those which depend upon traction methods are very thoroughly dealt with, and especial stress is laid upon the importance of semi-flexion of the joints in abolishing muscle tension. Every possible traction device is described and figured, but many of these are too complicated for general use. Mention of the use and value of the Thomas splint is conspicuously omitted, and this is a great defect in a book which aims at so comprehensive a survey of the subject. Operative methods and the use of bone-grafts are described much less fully than the traction appliances.

The second volume opens with a very notable article on fractures of the skull. This deals in great detail with the cerebral complications of head injuries. The physiology, etiology, and pathology of cerebral disturbances are described very well, and illustrated by excellent photographs, diagrams, and coloured plates. In this, as in other sections, practical details of treatment are not discussed at a length proportionate to that devoted to the more theoretical aspects of the subject. For example, we look in vain for any light on those types of fractured base which will be benefited by decompression; also there is no mention of traumatic defects in the skull or how they should be treated. The chapter on fractures of the jaw is well illustrated and practical. The figures showing methods of bone-grafting from the crest of the ilium to the jaw are particularly useful. Fractures of the spine and concomitant injuries of the spinal cord are discussed in the same way as are those of the skull. The coloured diagrams of nerve distribution are very useful, and the pathological preparations illustrating different forms of fracture and deformity are the most perfect we have ever seen.

The remainder of the work is concerned with individual fractures of the pelvis and long bones, and bears the same characteristics as those sections which have already been noticed, i.e., a great wealth of anatomical, pathological, and clinical illustrations, but a comparative paucity of description of modern methods of treatment, particularly in regard to the use of bone-plates, nails, or grafts. A list of references to the literature concludes the book. This appears to be lengthy and complete as regards German papers, but is quite inadequate in respect of French, American, and English work.

Practical Anatomy. By R. J. A. Berry, M.D., F.R.S.E., F.R.C.S.E. Second edition. 8vo. With numerous plates. (Vol. I, Superior and Inferior Extremities, pp. xiv + 472, 22s. 6d. net; Vol. II, Thorax and Abdomen, pp. x + 430, 22s. 6d. net; Vol. III, pt. 1, Head and Neck and Organs of Special Sense, pp. viii + 350, 20s. net; Vol. III, pt. 2, Central Nervous System, pp. viii + 256, 17s. 6d. net.) 1922. London: H. K. Lewis & Co. Ltd.

The second edition of Professor Berry's *Practical Anatomy* will be very welcome. It is now published in four parts, and is illustrated to a large extent by reproductions of the well-known plates of Ellis and Ford. Unfortunately the plates are reproduced without colour, and this is a very decided drawback.

The section which will be received best is undoubtedly that dealing with the "Anatomy of the Central Nervous System". The author claims, and justly, that the treatment of the subject differs materially from that given previously in anatomical manuals. The study of the gross anatomy of the central nervous system is combined with the study of its minute anatomy, its morphology, its functional importance, and its clinical applications. The author is to be congratulated very heartily on the large measure of success which has attended his efforts in combining so many aspects of the subject in one comparatively small volume. He has made the study of the central nervous system a very fascinating one, and has greatly increased its educational value to the medical student. His complete break with established tradition—so far as anatomical manuals are concerned—is an indication of the type of changes which are gradually being brought about in anatomical teaching.

The other volumes do not differ materially from the first edition. Some attempt has been made to omit minutiæ, and clinical applications of anatomy have been included, rather for the benefit of the graduate than of the medical student. The B.N.A. terminology is used throughout, except in the part dealing with the central nervous system; but the replacement of the common descriptive terms by those of a biological character is cumbersome, and is certain to be received, at any rate for some time to come, with disfavour in this country. It is difficult in certain parts of the body for the author to be consistent, and the term 'dorsal' is used with a double significance with reference to the calcaneus. On the whole one can hardly agree with the author that "neither the older generation nor the younger will experience any real difficulties with this thorny question of anatomical nomenclature". In this country anatomical nomenclature is still unsettled, and some general agreement, preferably amongst the English-speaking peoples, or at any rate in Great Britain, will be welcomed by teacher and student alike. As matters stand at present, the medical student cannot help but acquire a totally erroneous impression of the relative importance of anatomical terms and anatomical facts.

Traité Pratique de Cystoscopie et de Cathétérisme Urétéral. By G. MARION, Professeur agrégé à la Faculté, Chirurgien de l'Hôpital Lariboisière (Service Civiale), and M. HEITZ-BOYER, Professeur agrégé de Chirurgie des Voies Urinaires à la Faculté, Chirurgien de l'Hôpital St. Louis. Second edition, entirely remodelled. Large 8vo. Pp. 480, with 60 plates in black and white and colour. 1923. Paris: Masson et Cie. 100 fr. net.

THIS handsome volume, which first appeared in 1914, has been largely re-written and extended in the second edition.

There are four parts: (1) Cystoscopic examination; (2) Ureteral catheterization; (3) Cystoscopic treatment; (4) Cystophotography. The opening chapter deals with the instruments, and gives a short description of the optical system of the indirect cystoscope. The optical system which gives an inverted image has been used for most of the illustrations, and there is some discussion as to the comparative merits of the upright and the inverted images, which appears unnecessary, having regard to the complete disappearance for many years of the inverted image cystoscope. The preparation of the instrument and the patient and the technique of cystoscopy are described.

The body of the work is occupied by a description of the cystoscopic appearances seen in diseases of the bladder and kidneys. Of these, the chapters on cystitis and growths are the most detailed. The discussion of the cystoscopic diagnosis between simple and malignant tumours is not particularly helpful, and, with the wide experience of the writers, might well be extended. Less common diseases such as bilharzia, purpura, malakoplakia, and syphilis receive very short notice. The authors very properly sound a note of warning against the danger of cystoscopy in enlarged prostate. There is a short note on retrograde cystoscopy and on urethrocystoscopy and stereocystoscopy. The uses, limited in the authors' view, of the direct-vision cystoscope of the Luys type are described, and the application of this instrument in minor surgical interference in bladder disease is discussed.

Pyelography is described under catheterization of the ureters. The estimation of the renal function is discussed in a long and somewhat involved chapter. The urea concentration test is not mentioned, and the references to the work on this subject are entirely confined to the French literature. Lavage of the renal pelvis receives full discussion, and the authors favour solutions of nitrate of silver as the antiseptic for use. There is a good description of the treatment of bladder growths by the high-frequency current.

This is a very complete and useful book on cystoscopy and its varied applications. The illustrations are numerous, and those in colour are beautifully reproduced and realistic in effect. It will be of high value to the beginner, and also to those more experienced in the study of urinary surgery.

The Treatment of Fractures: with Notes upon a few Common Dislocations. By CHARLES LOCKE SCUDDER, M.D., Consulting Surgeon to the Massachusetts General Hospital. Ninth edition, revised. Royal 8vo. Pp. 749, with 1252 illustrations. 1923. Philadelphia and London: W. B. Saunders Co. 42s. net.

THIS book, which first appeared in 1900 and is now in its ninth edition, has evidently won deserved popularity in America. The present edition is an advance over its predecessor in that it includes many of the methods used or elaborated during the war, such as the Carrel-Dakin method of disinfection, transport and first dressings, and the use of the Thomas splint.

Proper stress is laid upon the principle of traction and suspension as the chief methods of treatment in recent fractures. Operative methods have been relegated so much to the background as to be almost out of sight. Otherwise all details necessary for treatment by splints are described carefully and with a great wealth of illustration.

Frakturen und Luxationen: Ein Leitfaden für den Studenten und den praktischen Arzt. By PROFESSOR DR. GEORG MAGNUS, Oberarzt der Chirurgischen Universitätsklinik, Jena. Royal 8vo. Pp. 87, with 45 illustrations. 1923. Berlin: Julius Springer. 3s. 4d.

THIS brief summary of the principles of fractures is intended as a guide for students. It is good and clear as an exposition of general principles, especially as regards treatment of fractures by Bardenheuer's traction methods; but it is too short in description of individual fractures and special methods of treatment to be of much value in these subjects.

Précis de Technique Opératoire: Chirurgie du Membre Inférieur. By GEORGES LABEY and JACQUES LEVEUF. Fifth edition. Pp. 248, with 280 illustrations. 1923. Paris: Masson et Cie. 10 fr.

THIS is an exceedingly neat little summary of operative surgery of the lower extremity, with illustrations which are ideal in clearness and finish. Osteotomy, operations on fractures, resections, arthroplasties, and orthopædic operations on the foot are treated well. The section on the operative treatment of fractures is almost limited to a description of French methods, those of Lambotte and Delbet having chief prominence. In transfixion of the lower end of the femur the transfixion pin is placed too near the knee-joint for safety. Parham's bands are represented as surrounding the conical lower end of the femur, where a flat band will not lie snugly. Operations on the neck of the femur and for exposure of the knee-joint are much too elaborate for a book of this type, whereas some simple methods are omitted. For example, transverse division of the patella and dove-tailing joining are described, whilst the vertical splitting method is omitted. One figure represents a drainage tube being taken right through the knee-joint in the way which ought to be avoided.

Apart from details of this kind, however, the book is a model of precise description and apt illustration. It does not include the subject of amputations.